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THE
LARYNGOSCOPE.

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OTITIS MEDIA WITH EFFUSION— A CHALLENGE TO OTOLARYNGOLOGY.*

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Otitis media with effusion is a common condition. Often present both in children and adults, it presents a challenge to otolaryngology. This challenge includes the etiologic, diagnostic and therapeutic aspects of this condition. It might even include the terminologic angle. It has been designated by many terms, none of which is satisfactory. An accurately descriptive term would be welcome. Otitis media with effusion is used in this presentation simply because it calls attention to the presence of fluid in the middle ear and not because it is an ideal term. To be certain, however, about the subject under discussion, it should be stated it is that condition in which the middle ear is filled with sterile fluid which may vary from a thin serous character (the most common) to a heavy, thick mucus mass. It is not related to the suppurative otitis medias except as it can be artificially created by the use of the biochemicals. The fluid may remain in the ear for only one day, or it may persist for many years.

There is abundant evidence that the diagnosis of this condition is often missed. Some personal experiences with over-

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looked patients in both Army and civilian life were cited when this subject was presented at the 1949 meeting of the American Academy of Ophthalmology and Otolaryngology and bear no repetition here. For years, teachers and textbooks alike have stressed, in the diagnosis of this condition a fluid level line. This is a snare and a delusion, for if one depends upon the observance of a fluid level line as a diagnostic aid in this condition more than nine out of every 10 cases will be missed. This is because the full tympanum and, much more often than not, the full mastoid as well, is 10 times as prevalent as the the ear which has only a level line. In none of our current textbooks, literature or teaching does one find these relative figures. It is not realized by most otologists and consequently is not taught to the younger ones.

Over the years in a general (not solely otologic) otolaryngologic practice, patients suffering with otitis media with effusion, percentage-wise, will total between 3 per cent and 4 per cent of all patients who visit the otolaryngologist. In view of this frequency and the fact that its diagnosis is often missed, the etiologic and diagnostic factors of this condition should frequently be reviewed.

ETIOLOGY.

Much is known of the etiology of otitis media with effusion but not all. It is the unknown which presents the challenge. When the unknown has become known we may have some therapeutic aids which are lacking now.

What do we know of the etiology of fluid in the middle ear? In general, it may be said that anything which will contribute to the Eustachian tube swelling and closure can be a contributing cause. More often than not an acute upper respiratory infection, abetted or not by the presence of local structural abnormalities or complicating factors, will precede the condition. These abettors are well known and require no detailed listing. (Dental malocclusion should perhaps be mentioned as it is sometimes overlooked.) If they are present, fluid is more apt to appear in the middle ear than if they are not; if one-sided, they may be the cause of the presence of fluid in the

homolateral ear; however, a blocked Eustachian tube is not, in itself, the whole answer to the problem, for if it were, every closed Eustachian tube would be accompanied by fluid in the middle ear. Why, then, when the Eustachian tube closes does fluid form in some ears and not in others?

Robison[1] has advanced the explanation that the presence or continuance of fluid in the ear is due to a blockage in the lymphatic glands which drain the middle ear region. Lately, our attention has been called to the sizable percentage among all who are afflicted with fluid in the ear, of those who are allergic. Jordan[2] puts it in the neighborhood of 70 per cent. Williams,[3] stating he believes the allergy in some to be physical in nature, intimates this additional type of allergy is present more often than is suspected. Both the regional blockage theory and the goodly number of allergic patients suggest that conditions outside the ear may be factors for the presence or continuation of fluid in the ears. It is in this area that we, as otolaryngologists, may have an Achilles heel. Focusing our attention on the ear and its neighboring parts, we may fail to recognize the presence of general conditions or disorders which may play a significant part in the condition under discussion. Anything which abnormally affects the water balance of tissues in the body can adversely affect the course of otitis media with effusion. Allergy, a known offender in this regard, has already been mentioned. Endocrine disorders of several types, not excluding pregnancy, are potential sources of trouble. Cardiac insufficiency and certain forms of cardio-vascular-renal disease have been known to be causative agents in the persistence of fluid. Hilger[4] has recently called our attention to autonomic nervous system disorders, many times psychic in origin, as disturbers of tissue fluid balance. All of these may underlie some of our cases of otitis media with effusion. Their possible presence challenges our diagnostic acumen. Unless we ferret them out, when they are present, we are doomed to a greater percentage of failure to cure than should be our lot.

Senturia[5] stresses the presence of a negative pressure in the tympanum as the mechanism by which fluid is drawn into

the ear. This undoubtedly is present in the instances where there is fluid but not all ears with a negative pressure develop fluid and, apparently, many have open Eustachian tubes long before fluid disappears from the ear. During World War II, when there were so many ruptured membrana tympani, an unusual opportunity was afforded for inspection of the middle ear mucosas. The majority were thin and dry. A small number, apparently without infection, were thick and moist. The cause behind the difference? One can speculate and run through a gamut of ideas from a poor development of the tympanal mucosa according to Witmaack's theory (advanced years ago) to the current emphasis on allergic mucous membranes. The cause of the difference, we can temporarily dismiss, but the fact that there is a difference, we must accept. This suggests that some ears under a given set of circumstances may develop fluid while others will not. Etiologically, the cause of the difference in mucosas does not matter, for when the patient presents himself fluid is already there. Therapeutically, however, knowledge of the cause of the difference may mean much, for that knowledge may hold the secret which will bring about an effective cure.

The two preceding paragraphs have stressed the possibility of a general condition as an etiological factor in the prevalence and persistence of otitis media with effusion. This must not be construed as an emphasis which excludes the local abettors mentioned above. Were it not for the latter, many of these "moist" ears would never develop fluid. That local pathology can be etiologic and may be a cause for persistence must never be forgotten. In protracted cases, for instance, one must repeatedly look at the nasopharynx lest a developing malignancy be overlooked.

Eagle[6] and Tobey[7] in recent years have published reports which mention a recent marked increase in the incidence of otitis media with effusion. Politzer[8] described the condition accurately and wrote at length of it in his textbook, which suggests it was well known and often recognized in the latter part of the last century. The present author has noted only a 20 per cent increase in the incidence of fluid in the ear since

the advent of the biochemical treatment of purulent otitis media. (With the use of this therapy and consequent disappearance of pus cells, the ear can look and act like one which has never gone on to suppuration.) The greater part of the 20 per cent increase is to be found in children. It goes without saying that there must be thousands upon thousands of such ears which never come to the attention of the otolaryngologist, for few pediatricians or general practitioners test the hearing following the use of the biochemicals for suppurative otitis media, and fewer would recognize the presence of the condition if they happened to look into the ear.

Fluid in the ears may develop after trauma or in aerotitis. These cases are few in number and do not present the problem of persistence in like degree to those caused by other etiological factors.

The effusion has been known to continue in its fluid state and remain present in an ear for years. Sometimes after one, or repeated attacks (and these occur), there are resultant adhesions which bind the drum and ossicles to each other and the promontory, with a consequent hearing loss which varies with the location and strength of the adhesions. Why some ears will clear in a reasonable period, others have a persistence of fluid for months or years, and still others terminate with adhesive processes is not exactly known. As we learn more about tissue chemistry, general metabolic processes, electrolytic balances, etc., we may arrive at the final answers.

PATHOLOGY.

There are two types of fluid which may fill the ear. One is of a thin serous character and a negative pressure in the middle ear is the cause of its presence. It is a transudate. Lowy,[9] Robison and others have pointed this out. The second is mucus in character and is exudative. When present, it is usually mixed with the serous transudate. The degree to which mucus is present is indicative of the mucosal response to inflammation. The amount, as intimated, is variable. There may be none and it is conceivable the entire fluid response of an ear may be mucus in character. While there may be many

variations of the mucus contents of the fluid, clinically an ear is either a "serous" ear or a "mucus" ear, the former having a preponderating incidence. Both may persist for weeks, months or years and to state that one is acute and the other chronic is erroneous. Once the initial response has occurred, a change from a serous to a mucus ear, or vice versa, is relatively uncommon. Uncommon though it may be, the change from the serous to the mucus is the more frequent. There is a high protein content of the serous fluid with excessive coagulability when exposed to air. The mucus fluid has a low protein content. In either event, the fluid is sterile. Many investigators have tried but have failed to demonstrate the presence of any of the pathogenic respiratory bacteria.

One cannot stress too strongly the fact that the process is one which involves the entire middle ear and not the tympanic cavity alone. The middle ear is a continuous anatomical structure from the mouth of the Eustachian tube to the last cell in the tip of the mastoid and the condition under discussion involves the mucosa of the mastoid cells as well as the mucosa of the tympanic cavity. This fact accounts for the rapid reappearance of fluid after removal of some from the tympanic cavity by mechanical interference. X-rays of the mastoid will give suggestive evidence that this is so, and operation on the structure does reveal the presence of fluid in the cells. Jervey,[10] Lawson,[11] Cody,[12] Tobey and others have testified affirmatively in this regard.

HISTORY.

A typical history will usually tell of a partial loss of hearing in one or both ears following a cold, usually accompanied by a tinnitus and the symptom of autophony. If there is air as well as fluid in the tympanum the patient may complain of "bubbles" in the ear, or state simply that at times there is loss of hearing, at other times none, depending upon the position of the head and the consequent movement of fluid in the ear. If the tympanum is filled with fluid there will usually be a complaint that the ear and head on that side have a "blocked feeling" and that the hearing is poor in the affected

ear. In a fair percentage, the story of a previous cold cannot be elicited. It is well to remember that the disease can be and often is bilateral.

DIAGNOSIS.

The diagnosis is usually made by inspection; sometimes has to be made by auscultation; and, occasionally, can be made only by assumption which can then be supported by a diagnostic paracentesis.

Examination in the inflammatory stage shows a dilatation of the blood vessels of a drum whose landmarks are still present. If a conductive hearing loss is present which is somewhat out of proportion to the amount of pathology seen, it should make one suspect the presence of fluid. At a slightly later stage, the observation of a meniscus makes the diagnosis easy. Bubbles of air, too, when present in the fluid give the drum a characteristic appearance. If there is doubt about the existence of bubbles or a fluid level line, the use of the pneumatic otoscope can be of material diagnostic aid.

More difficult to diagnose is the ear, the tympanic cavity of which is completely filled with fluid and the only identifying feature is the overall amber color of the drum. The amber color can vary from a faint, barely perceptible, yellowish tinge to a dark, almost bluish cast approximately the color seen in the hemotympanum, yet even in the latter the amber tint is perceptible. This is the picture most frequently missed diagnostically. A helpful aid is the fact that in almost all of these drums the short process and the handle of the malleus stand out as chalky white in appearance, in contrast to the colored drum. This chalky white appearance of the short process and the handle of the malleus gives one the impression that the drum is retracted when, as a matter of fact, it may not be at all. The use of the pneumatic otoscope often shows up a wrinkled appearance to the drum, a finding not frequently found otherwise. In a majority of the ears which contain fluid of long standing, the drum, with the use of this instrument, will be found to be flaccid. In many of these, the drum will have a translucent appearance and look as though

it were made of oiled paper. The picture, depicted above, variable as it may be from a light to a dark colored drum and thus somewhat confusing until readily recognized, represents the full tympanum (and probably the mastoid as well), and should be found 10 times as frequently as the drum with a meniscus. Until one reaches this diagnostic percentage more practice is needed in the examination of these ears.

Auscultation can be of help but is not needed for diagnosis if the amber color is recognized. The auscultatory sound is necessary for the diagnosis of fluid in the ear if an opaque drum is present and otitis media with effusion does occur in ears, the drums of which are opaque. The diagnosis of fluid in the ear is far more easily overlooked in such ears, for the loss of hearing present is usually assigned to the thickened drum (with possible adhesions).

There are a variety of auscultatory sounds. If there is some air as well as fluid in the tympanic cavity there is a resulting bubbling which denotes the presence of fluid. One never hears an auscultatory bubbling unless the tympanum has air as well as fluid in it. If the tympanum is completely filled with fluid, entirely different auscultatory effects are heard. The most usual is one which is like the chug of fluid heard when a large bottle partly filled with a heavy fluid is shaken. Even this description is inadequate. It is a difficult sound to recognize and interpret. Often it is heard only on the first auscultatory blow and subsequent blows will produce no sounds suggestive of fluid. Unless one listens carefully for the first blow, the diagnosis may be missed entirely. Occasionally one will hear a faint crackling immediately after the termination of the blow. Also, occasionally one hears only a roughened auscultatory sound, with no suggestion of fluid in it and the comparison with the auscultation sound in the well ear is needed to appreciate that there is something unusual in the affected ear. The necessity for making these discriminations is the reason why careful, gentle catheterization is needed, for the sounds which must be heard are clearly discernible only when very gentle inflation is practiced. It can easily be missed by a loud or forceful blow. The crude blow

of politzeration, with its attendant extraneous sounds, may miss the desired one entirely. Infrequently, though certainly, one hears no sound at all and one wonders if the tube has been catheterized, but inspection of the drum in these instances will show bulging as compared to a precatheterization, normal contour.

The diagnosis of fluid in the ear can and should be aided by the employment of diagnostic paracentesis of the membrana tympanum. The use of paracentesis is certainly diagnostic and probably therapeutic as well. Its therapeutic employment will be mentioned later. The procedure is identical whether it is used for one or the other purpose.

Paracentesis can be done with a hypodermic syringe needle or a myringotomy knife. A majority of those who use paracentesis prefer the latter. In almost all instances a small opening (the width of the myringotomy blade) just anterior to the annulus at six o'clock will suffice. With a surgically clean myringotomy knife it can be done without anesthesia (even in children) and without sterilization of the canal, with no untoward results and with but little pain to the patient. It is wise to precede this with inflation. This will place the drum further from the inner tympanic wall than usual. The puncture will be less painful if the inner wall is not touched. If inflation has preceded the puncture, in most instances some of the fluid will flow into the external canal immediately following the removal of the knife from the drum. This will not represent all of the fluid in the tympanum and the removal of the remainder should be accomplished by further inflation or the use of suction. With inflation, the fluid, except in ears with a high mucus content, runs freely into the canal and out into the hollow of the concha. It has been called straw colored by many. No matter what name is given, it is this yellowish or amber colored fluid which gives the drum its characteristic appearance and whether the change in color of the drum in otitis media with effusion be light and barely perceptible, or dark and almost bluish, there will be the underlying amber to all except the opaque drums.

Suction with the use of the pneumatic otoscope (Siegle) which has been advocated following paracentesis and which creates a negative pressure seems to be contraindicated. Its use apparently aggravates the condition. This method does not remove as much secretion as does inflation or "spot" suction for much of the fluid drawn into the canal when the negative phase is used returns to the tympanic cavity when the negative pressure is released. The writer has tried this and with dismay has watched much of the fluid return through the paracentesis opening into the tympanic cavity when it was thought all of the fluid had been captured for the patient's relief as well as his. There are some who decry the use of inflation for the evacuation of fluid after paracentesis and urge only spot suction. The writer has employed both and can find no difference between them.

That there is a negative pressure in the ear at times can be learned by watching the drum following paracentesis. Very often a rising bubble can be seen as the negative pressure draws air into the tympanic cavity up through the fluid. Occasionally, following paracentesis, several bubbles of air can be seen doing this. On occasion, too, after a paracentesis and the removal of fluid by spot suction, one can see the drum change from its preparacentesis amber color to the usual, normal gray appearance, only to have the tympanum, seen by continued inspection of the drum, refill with fluid with a concomitant return of the amber color. This picture of a rising fluid level line is an intriguing one. The tympanum, under continued inspection and appropriate timing of spot suction, can refill as many as three times at one sitting. This is quite conclusive proof that the fluid is greater in amount than can possibly be contained in the tympanic cavity.

If the fluid in the tympanum is of the heavy mucus type, it may be necessary to employ additional measures in order to remove it from the ear. Often, in this type, it is necessary to make two openings in the drum, one an incision at the site of the usual paracentesis and another, a paracentesis, in the superior portion of the drum, preferably in the anterior portion. A heavy mucus secretion usually comes out in one mass.

On such occasions, unless the second opening is made by the time one-quarter, one-third or one-half of the mucus mass has been suctioned into the canal, the negative pressure in the tympanum is so great that the remainder cannot be suctioned from the cavity. On rare occasions it may be necessary to have an assistant inflate the ear while one is employing suction in order to remove all of the contents of the tympanum and give hearing relief to the patient. It is in this mucus type of otitis media with effusion that, with the employment of suction, one often sees a strand of mucus pulled two or three inches out through the paracentesis opening and into the canal by the suction tube. The color of the mucus has less of the amber tint than the serous fluid. Fortunately, these cases, which are more difficult to handle, are in the minority. If, after paracentesis and removal of some fluid, the patient reports the ear still feels dull or that the hearing remains dull, he is more than likely correct and one had better re-examine the ear and ascertain if there is not more fluid present. There well may be more fluid, particularly if it is of the heavy mucus type.

The paracentesis opening closes all too readily. In the great majority, if conscientious attempts have been made to remove all the fluid possible, no further fluid will escape. Occasionally some will continue to escape for a few hours and rarely the ear will drain for a day or so.

It may be argued that without sterilization of the canal, paracentesis may be dangerous on account of possible infection and subsequent suppuration. Having performed about 2,500 paracenteses, the writer has seen but one go on to suppuration and this was in a patient who had the very onset of a concomitant cold. The ear was dry and the hearing had returned to normal within a fortnight.

Apparently many paracenteses can be performed without subsequent injury to hearing. Hunt[13] has reported he has done over 80 in one ear, that of a doctor, with eventual cure and normal hearing. While few, if any, have made as many paracenteses as this, there is abundant evidence, if properly done, there is no real damage to hearing.

There is no characteristic hearing curve, for the pathology under consideration may and often does occur in ears already diseased. Pre-existing inner ear disease may also be present. In an uncomplicated ear, the presence of otitis media with effusion will, of course, give the picture of a conductive deafness. Usually the deafness is out of proportion to the pathology seen on examining the ear, though, on occasion, the fluid, small in amount, is in such position that it does not block the conductive pathways and the hearing is quite good. This, of course, is infrequent because in the great majority of these ears the tympanum is entirely filled with fluid.

It should not be forgotten that the diagnostic endeavors should be directed not only to the ear but to the discovery of general conditions which are abnormal.

PROGNOSIS.

It has been stated previously that fluid in the ear may remain for only a single day or it may persist for years. It is rarely possible to predict with certainty which ear may be fortunate and which may not. In general, it can be said that the ears with the greater amount of mucus content will present more of a therapeutic problem than the others. It must be remembered, however, that a serous fluid can persist in the ear for years. The smaller the amount of fluid in the ear the more likely there will follow a rapid recovery. If fluid has been present in the ear for a lengthy period before treatment is instituted, a protracted course is much more likely than if its presence is of short duration. Undiscovered, or untreated, complicating factors may result in persistence of the fluid. Repeated attacks, which occur with a moderate degree of frequency, suggest unrelieved local or general pathology. If the condition can be cleared, the hearing in almost every instance returns to its former level. Unless subsequent attacks produce adverse conditions, the ear apparently is as good, functionally, as before. On the other hand, if the pathological state persists, eventually changes take place which are detrimental to function and these are irreversible.

TREATMENT.

The treatment for otitis media with effusion is not specific and consequently challenging. In the early stages, therapeutic endeavors should be directed toward elimination of a respiratory infection if one is present. When the infection is over, treatment can be directed to the ear. Without antecedent respiratory infection, the ear should receive immediate therapeutic attention. Politzeration or catheterization may be employed once, or a few times, and in many instances this is all the treatment required. If the condition does not clear with these manipulations, attention should be directed to local and general pathologies or irregularities. At the same time, or later, according to the inclination of the attending otolaryngologist, therapeutic paracentesis with subsequent removal of fluid by politzeration, catheterization or spot suction may be employed. Direction of attention to local or general complicating factors may call for tonsillectomy or adenoidectomy, removal of secondary adenoid growth, correction of nasal abnormalities, X-radiation for lymphatic hypertrophy or regional lymphatic blockage (employment of radium applicators to the nasopharynx in particular has not been demonstrated to be of worth; Jordan has noted this), correction of dental malocclusion, allergic management, endocrine therapy, treatment for cardiac insufficiency, etc.

Thereapeutic paracentesis is decried by some, praised by others. The writer employs the procedure, even with the knowledge that it is not all sufficient because of the overwhelming relief that it affords the patient, for which the latter is so grateful he is willing to continue treatment even though it be protracted; because the majority require only one paracentesis for recovery and because the longer the fluid is present in the ear before treatment is instituted the longer the time required to effect a cure. Therapeutic paracenteses are carried out at varying intervals, according to the symptomatology of the patient, coincidental with other measures, local and general. It is considered that the employment of both is wise, as the use of one without the other is not so effective in affording a cure. As a last resort when all other

measures have failed, a very small number may require mastoidectomy before relief can be obtained. If it is employed it would seem wise that the modern technique of a "closed" postoperative wound would be abandoned and the employment of an acrylic or tantalum tube extending from the antrum to the skin be substituted. The tube should be left *in situ* with the use of general biochemical coverage to avoid infection until evidence is present that fluid is not re-forming and, with "closure," there will be no return of it. A very few ears, despite many forms of therapy, will have a persistence of fluid. It is probable that these ears represent undiscovered complicating factors.

COMMENT.

This presentation is written with the hope that it may stimulate an increased interest in a wider recognition of otitis media with effusion. The mere printing of these words will not accomplish this. Introduced more than four months later to an otolaryngologist who listened to the verbal presentation of this subject at the last Academy meeting, the writer interestingly asked, "Have you diagnosed any cases?" The honest answer was, "I'm afraid I have not." That there would not be one case in an average otolaryngologic practice during a four months' period is incredible. No, words will not suffice. If ever this condition is to be widely recognized, it must be accomplished by personal demonstrations of the actual presence of fluid in the ear, particularly the ear with the full tympanum. This, in turn, must be done by those who have been privileged to have known teachers who have demonstrated it to them.

The challenge of otitis media with effusion remains. More effective teaching of this subject is required by those in a position to do so.

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ARYTENOIDECTOMY.
A PRELIMINARY STUDY.*

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Bilateral vocal cord paralysis following subtotal thyroidectomy occurs with some frequency. To correct the deformity, operative procedures were attempted with the hope of obtaining outward rotation and displacement of the arytenoid cartilage, resulting in an increase of the laryngeal airway and maintenance of an adequate speaking voice. King[1] first developed a satisfactory operation for the correction of bilateral midline vocal cord paralysis. Kelly[2] modified the procedure by removing the arytenoid through a "window" in the thyroid cartilage. Utilizing the basic Kelly principle, Woodman[3] simplified the approach by separating the cricothyroid articulation and elevating the posterior border of the thyroid cartilage.

Although the effects of chronic laryngeal obstruction upon the physiology of respiration and circulating red cell mass are known, it was felt that the changes recorded before and after successful arytenoidectomy for post-thyroidectomy bilateral abductor laryngeal palsy would be of general interest.

METHODS.

Spirometric tracings were obtained, giving tidal volume, vital capacity and maximum respiratory capacity. The laboratory procedures included routine red blood cell counts, hemoglobin determinations by the Kleb method, and calculated cell

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indices. Vital capacities were also done with a modified spirometer; chest measurements were made at the level of the fourth interspace before and after arytenoidectomy. All studies were done under standard hospital conditions and at rest. Postoperative studies were made at intervals ranging from 24 to 36 weeks.

RESULTS.

Fig. 1 is the preoperative photograph of the patient in Case 1, a 33-year-old-female on whom a thyroidectomy was

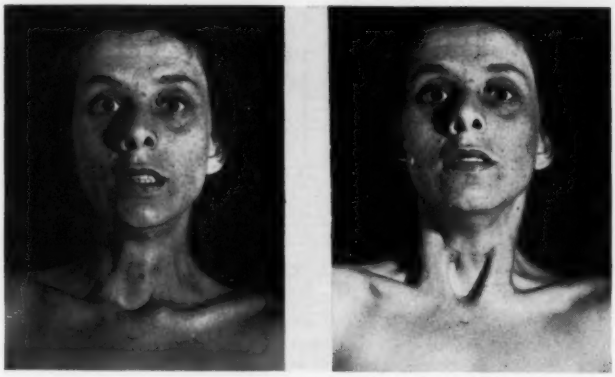

Fig. 1. Case 1, 33 years old, showing prearytenoidectomy respiration. (A) Quiet respiration. (B) Forced inspiration.

done 14 months prior to this study. Immediately after thyroidectomy she noted the inability to talk above a whisper, and following ambulation she noted increasing dyspnea upon exertion and orthopnea at rest. This was associated with palpitation and a marked inspiratory stridor. These photographs depict the utilization of most accessory muscles of respiration, both during deep inspiration as well as during quiet inspiration.

Fig. 2 represents the prearytenoidectomy spirometric tracings in the same case. There were predominantly sighing respirations occurring at approximately every 40 seconds, with accompanying laryngeal stridor. The actual rate of

breathing averaged 12 respirations per minute and the tidal volume approximately 500 cc. The complemental sighs were approximately 1,700 cc. Vital capacity averaged 2,700 cc.

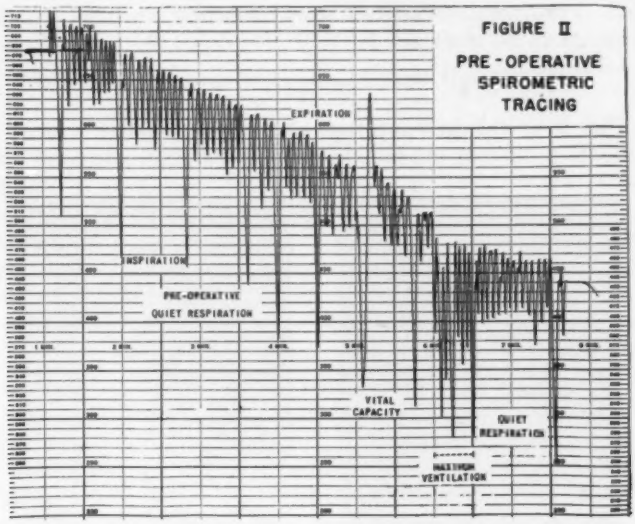

Fig. 2. Prearytenoidectomy spirometric tracings, Case 1. Note sighing respiration, slow respiratory rate, and low maximum ventilation.

The minute respiratory volume was 7,700 cc. The maximum ventilation per minute was 8,800 cc. (approximately 10 per cent of normal), and the maximum rate was 15.

In contrast, Fig. 3 represents the postarytenoidectomy photographs of the same case, and it is obvious that the quiet respiration as well as the deep respiration was improved. Both the intrinsic and extrinsic laryngeal muscles were very much enlarged at operation. The postoperative spirometry, as seen in Fig. 4, demonstrated the absence of deep, sighing respirations; and, although not demonstrated, the absence of stridor. More dramatic, however, was the increase in rate to the average of 18 per minute with a tidal volume of 400 cc. and a minute volume of 6,000 cc. Although there was little

change in the vital capacity, the postoperative maximum respiratory capacity revealed an increase of approximately 31 per cent (8.8 to 12.7 liters).

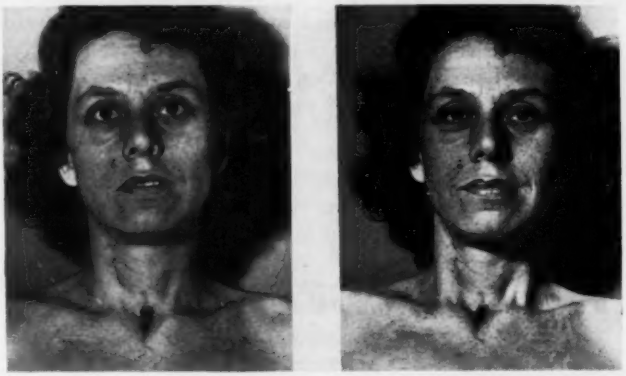

Fig. 3. Same case as Fig. 1. Postarytenoidectomy respiration. (A) Quiet respiration. (B) Forced inspiration.

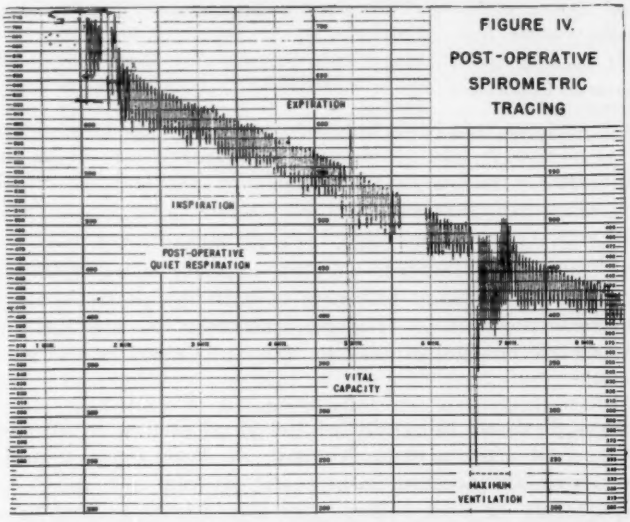

Fig. 4. Postarytenoidectomy spirometric tracings, Case 1. Note increased respiratory rate, absence of sighing and increased maximum ventilation.

Case 2 was a 56-year-old female whose thyroidectomy was done 12 months prior to study. Immediately following thyroidectomy there was a change of voice and dyspnea, palpitation and orthopnea were apparent on ambulation. Case 3 was a 65-year-old woman whose history of post-thyroidectomy bilateral palsy dated six years prior to her arytenoidectomy. Increasing dyspnea on exertion existed postoperatively with associated inspiratory stridor and palpitation. No spirometry was done on these cases.

It may be seen from Table 1 that in all cases there was some decrease from the theoretical normal in vital capacity and chest expansion prior to arytenoidectomy with an asso-

TABLE 1.

	Preoperative			Postoperative		
	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3
Respiratory Rate (per min.)	12	13	14	18	16	16
Tidal Volume (cc.)	525			400		
Minute Volume (cc.)	7,700*			6,000		
Vital Capacity (cc.)	2,700	1,000	1,600	2,500	1,800	1,800
Maximum Respiratory Capacity (cc.)	8,800			12,700		
Chest Expansion (cm.)	2.54	1.27	4.44	5.72	3.81	5.72

*Including compensatory sighs.

ciated slow rate of breathing. In contrast, although there was little significant increase in vital capacity after operation, there were significant increases in both the chest expansion and respiratory rate. It might, therefore, be assumed that spirometric changes similar to those in Case 1 could be expected.

Table 2 shows the result of the blood studies in all cases before and after arytenoidectomy. In both Cases 2 and 3 there was an increase in hemoglobin, total red count and hematocrat. In Case 1 there was a relatively normal hemoglobin and red count, although the hematocrit was slightly

TABLE 2.

	Preoperative			Postoperative		
	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3
Hemoglobin (grams)	13.8	16.4	16.4	13.0	13.8	13.6
R. B. C. (millions per cm.)	4.24	5.44	4.71	4.54	4.87	5.9
Hematocrit (per cent)	47.0	50.5	51.0	46.0	45.0	47.0
M. C. V. (cubic microns)	112	93	106	101	93.7	80
M. C. H. (mm. grams)	33.0	30.5	34.8	29.0	28.7	23.0
B. P.	110/80	130/80	184/90	126/86	138/88	150/90
Pulse	100	90	80	100	80	70

elevated. In all cases the mean corpuscular volume was increased preoperatively, whereas the mean corpuscular volume and the mean corpuscular hemoglobin decreased. There was little change in the blood pressure, although there was some decrease in the resting pulse rate. Symptomatically, all patients showed definite and marked improvement in physical endurance. Their voices improved, stridor ceased and all were able to sleep without extra pillows.

DISCUSSION.

From these studies, it becomes apparent that arytenoidectomy performed by the method of Kelly and Woodman is a logical procedure for relieving bilateral abductor laryngeal palsy; furthermore, marked interference with the exchange of air is improved, as evidenced by the improvement in maximal respiratory exchange as well as by the improvement in thoracic expansion and respiratory rate. That compensatory mechanisms for the maintenance of normal tissue respiration are utilized is reflected by the relative increase noted in the preoperative blood cell indices. Postoperatively, these studies indicate that there is a tendency for the secondary or compensatory blood changes to return to normal values; furthermore, it is suggested that a means through which more oxygen is transported to the body is by relative increase in cell volume as well as hemoglobin concentration and not entirely through increase in the total number of red cells.

SUMMARY AND CONCLUSIONS.

From these preliminary and comparative respiratory studies of three cases representing post-thyroidectomy bilateral laryngeal abductor palsy treated by arytenoidectomy, it is apparent that:

1. The marked interference of the exchange of air accounts for the symptoms and evidences of chronic hypoxia.

2. The procedure is a practical means of correcting the interference in ventilation and preserving adequate voice.

3. After operation there is a definite return toward normal of the respiratory exchange of air as evidenced by the increase in respiratory rate, elimination of sighing respirations and stridor, the increase in maximum respiratory capacity; furthermore, a return towards normal of the red cell indices show this same trend.

4. It is suggested that in older patients the increase in actual cell size and hemoglobin is a compensatory mechanism which aids normal oxygen transport to the tissues (Cases 2 and 3).

5. It appears that in younger and more vigorous individuals the principal compensatory mechanism is an increase in ventilation (Case 1).

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NEW PILLING STORAGE BATTERY FOR E.N.T. INSTRUMENT LIGHTING.

Apparently the battery troubles of bronchoscopists and other physicians using battery-lighted instruments are over. George P. Pilling & Son Co., of Philadelphia, Pa., one of the country's oldest and largest surgical instrument houses, has just announced a new, portable, practically foolproof storage battery unit that is both powerful and dependable. What's more, it weighs only 16 pounds and can be easily carried around by even the smallest O.R. nurse.

The unit contains two 4 volt, 6 ampere nonspill storage cells in transparent plastic cases. Only one is used at a time, leaving the second always in reserve. A rheostat allows instant regulation of the brightness of the lamps, and a quick change from one battery to other by the flip of a switch does not require any changes of the instrument light cord tips. The condition of each battery may be determined by a glance at the charge indicator. And recharging is simple with the built-in charger. Just plug in the regular house current if it is the usual 110-115 volt, 60 cycle A.C. Descriptive literature is available.

HEAD AND NECK PAINS OF CERVICAL DISC ORIGIN.*

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(by invitation),
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Headache is the most common human affliction and for centuries has clinically been associated with intracranial pathology. Most textbooks treat it solely as a symptom manifesting disturbed cerebral physiology, yet for almost 200 years the cervical origin of headaches has been suggested. Wolfe, in his excellent monograph, "Headaches and Other Head Pain," devotes less than a page to the neck as a source of headache. Other monographs often omit discussion of headache of cervical origin or attribute it to myositis, arthritis, spondylitis, tumors, Potts' disease and similar lesions. An abundance of evidence has accumulated so that we should consider the neck as one of the primary sources of head pain. Almost every neurosurgeon and many otorhinolaryngologists have had patients experience frontal, temporal or facial pain when the occipital region or neck was injected with local anesthetics. In almost all head injuries, trauma to the neck is incurred, and we now believe many of the post-traumatic headaches are due to cervical injuries. It is noteworthy that post-traumatic headaches are much less frequent following penetrating gunshot wounds than after blunt injuries to the head where the possibility of neck injury is greater.

One of us (R. E. S.) has observed cervical discs for many years and has always been impressed by the frequency of associated headache and facial pain manifested by these sub-

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jects. Such pains are often the earliest manifestation of cervical disc pathology before the development of radicular symptoms occurs. In reviewing our own patients with cervical disc syndromes, we have found headache to be a prominent symptom in more than 50 per cent of those not requiring surgery. Among patients with severe pain in the upper extremity requiring surgery, less than 25 per cent list headache as a symptom.

Symptoms: Headache of cervical origin presents a rather typical clinical picture. The pain is chronic, recurrent and usually begins in the suboccipital region on either side of the head. After a few hours, the pain radiates anteriorly to the homolateral eye, forehead, temporal and occasionally maxillary regions. Usually described as a "dull ache or nagging pain," it may at times be throbbing in nature. This is especially true when the pain is retro-orbital. A common complaint is "it feels as if my eye is being pushed out." Such pain may last for several hours to several days. Rarely, patients have had constant pain for weeks when they are first seen. At times the pain is lancinating in character. Beginning in one suboccipital region, it may last for minutes or hours, only to disappear, leaving a residual soreness of the involved areas. Several such episodes may occur in a day, or days may intervene between attacks. At times pain may occur in only one region of the head or face, excluding all others. Thus, patients with only severe frontal, orbital, maxillary or temporal pain are frequently encountered. Occasionally, the homolateral eye, when painful, will water. We have never observed watering of the nose in this syndrome.

Examination of such patients may show remarkably little, especially if it is early in the course of the disease. Upon close questioning, one is able to obtain a history of neck injury, recurrent stiff or painful neck, or shoulder and upper extremity pain in a high percentage. Tenderness is usually present over the supra- and infraorbital nerves when pain in the frontal and maxillary region has been prominent. Likewise, pressure over the greater and lesser occipital nerves in the suboccipital region produces *extreme* pain. The brachial

plexus of the homolateral side may also be extremely tender on palpation. When, on pressure over the occipital nerves, the brachial plexus or head (to compress the cervical spine) results in duplication of the patient's pain, we feel this finding to be highly suggestive, if not pathognomonic, of cervical disc disease.

Such individuals usually have a straightening of the cervical spine, the normal lordotic curve being lost. The posterior cervical muscles are spastic and often tender. Without realizing their neck is the source of trouble, some patients keep their head tilted slightly to one side, having observed that quick or excessive motion of the head results in severe head or face pain. That position of the head is often an important factor is exemplified by a recent patient who stated that his pain came on only following dinner. It was his custom to sit in a deep chair to read the evening newspaper. His position was one of acute flexion of the neck and, after a short period of time, this resulted in acute frontal and orbital pain lasting for hours and requiring opiates for relief. It is noteworthy that, although manipulation of the head may produce head or facial pain, traction on the head for a few moments does not give the same measure of relief it does in shoulder pain due to cervical disc ruptures. Likewise, pressure over the cervical spinous processes is less apt to cause head and face pain than shoulder pain.

In the diagnostic study of patients with chronic, recurrent headaches, X-rays of both the skull and cervical pain should be obtained. In the absence of clinical findings indicative of intracranial pathology, X-rays of the skull are of little help. Cervical spine studies reveal in early cases merely loss of normal cervical lordosis, while in more advanced cases intervertebral space narrowing, reversal of the cervical curve, hypertrophic lipping and ligamentous calcification are encountered.

Mechanism of Pain Production: The mechanism by which headache or facial pain due to lesions of the cervical discs are produced is unknown. Spasm of the posterior cervical muscles with resultant traction on the frontalis muscle has been

used as explanation for pain of the forehead and orbit. Since tenderness of the supraorbital, infraorbital and occipital nerves is often present, a neurogenic source of the pain is suggested. Anatomically, the dorsal horn of the upper cervical nerves (*i.e.*, the sensory portion of these nerves), termed the substantia gelatinosa of Rolandi, emerges imperceptibly with the spinal tract of the Vth cranial nerve. Thus, theoretically, noxious impulses entering the upper cervical nerves may be referred over the distribution of the trigeminal or upper cervical nerves. Pain of lancinating character is more readily understandable by this theory. Further credence is lent the neural origin by the complete relief of pain often afforded these patients when very small amounts of 1 per cent novocaine solution are infiltrated around the occipital, supra- or infraorbital nerves.

Differential Diagnosis: The presence of chronic, recurrent frontal or maxillary pain leads many of these patients to seek the help of an otorhinolaryngologist. Indeed, many of our patients are referred to us by them when the sinuses are found to be free of disease. The chronicity of the pain, absence of infection, and normal appearing Roentgenograms, among other findings, readily excludes the sinuses as the source of pain. Trigeminal tic douloureux is often suggested when the upper two-thirds of the face is the painful area. True trigeminal neuralgia can usually be excluded by a careful history and always by witnessing an attack of pain. The pain of tic douloureux is lightning-like, momentary and excruciating. Several such episodes may recur in rapid succession. So-called trigger areas which, when stimulated, reproduce typical attacks are present. Cervical disc pain referred to the face is usually of many hours' duration and trigger areas are never present. The term atypical facial neuralgia is often applied to pain of this nature.

Migraine and histamine cephalgia are probably the two most frequent diagnoses encountered in patients with cervical disc lesions. The term migraine is far too frequently used to explain unilateral head pain. In a strict sense, it should be reserved for those individuals with recurrent, throbbing

headache, usually unilateral at onset, accompanied by nausea, vomiting and irritability at the height of the attack and preceded by visual phenomena such as blurred vision, scintillating scotomata, photophia or hemanopia. Less frequently, speech disturbances, paresthesias and vasomotor disorders are encountered. A family history of migraine is usually obtained. Ergotamine or Dihydroergotamine 45 parenterally affords prompt relief of migrainous headache. The term histamine cephalgia on the other hand should be restricted to chronic, recurrent unilateral pain over the distribution of the branches of the internal and external carotid arteries, accompanied by vasomotor disturbances such as profuse watering and congestion of the eye, rhinorrhea, nasal obstruction, increased perspiration and frequently flushing of the skin. Such an attack may be precipitated by injection of 0.35 mg. of histamine base. Intravenous epinephrine in 1:400,000 dilution promptly terminates an attack.

Treatment: Since we believe the facial and head pain occurring in cervical disc lesions results from nerve root irritation, treatment is largely directed toward mechanically diminishing or preventing nerve compression. In the milder cases, merely cautioning patients against rapid or exaggerated head motions, the omission of a pillow during sleep and the use of heat in the form of a hot water bottle beneath the neck is all that is necessary. Among those with severe headaches, cervical traction affords satisfactory relief. Our routine is to hospitalize these patients for several days to acquaint them with the proper use of the traction apparatus. Five pounds of traction is applied with the usual cervical halter. Except for mealtimes and bathroom privileges, the traction is constant until the acute pain subsides. Gradually they spend more time out of traction when they are discharged to their homes and instructed to use traction only at night. The average hospital stay is about one week. Local anesthesia may be employed with considerable benefit in patients with severe headache associated with occipital nerve tenderness and in acute facial pain over the distribution of the supra- and infraorbital nerves associated with exquisite tenderness of these structures. A few cubic centimeters of

1 per cent novocaine infiltrated in the region of the occipital, supraorbital or infraorbital nerves often produces prompt relief of discomfort for hours. Longer acting anesthetic agents such as 2 per cent metycaine prolongs the relief of pain. When a considerable quantity of procaine is used, its systemic effect may give temporary relief.

Case Report: The following is a representative case of headaches due cervical discogenetic disease. C. L. W., age 56, the father of two physicians, was seen on Nov. 11, 1949. One week previously, pain occurred in the left occiput and radiated to the left eye. It was throbbing in nature, and constant, lasting about six to eight hours. Occasionally sharp, stabbing pain occurred in the eye. Although opiates were administered, pain relief was incomplete. One or more such attacks occurred daily. During the past 48 hours he had had no relief of pain. On questioning, he admitted previous mild neck discomfort, frequent cracking sensations in the cervical region but denied any previous neck trauma. Neurological examination was negative except for the following: The head was tilted to the right, and the normal cervical lordosis was lost. The posterior cervical muscles were in spasm and neck mobility was reduced. Compression of the head increased his symptoms, while traction for a few moments diminished them. Pressure over C-5 and C-6 spines, as well as momentary compression of the neck veins, increased the headache. The blood pressure was 160/100. There were no reflex or sensory changes. Our Roentgenologist reported "examination of the cervical spine shows narrowed intervertebral spaces at the fifth and sixth levels with some proliferative spur formations along the anterior margins and in the anterior ligaments. Very short nub-like spurs project into the foramina at the same level."

He was placed in cervical traction, using five pounds of weight In 12 hours the pain was markedly diminished and in 48 hours it was gone. The duration of traction was reduced daily so that after one week he was using it only at night. Pantopaque myelography was carried out on the seventh hospital day. Spinal fluid dynamics were normal. The fluid contained no cells and 56 mg. per cent protein. Myelography revealed failure of the cervical nerve root sacs to fill on the left at C-5 and C-6 levels. He was discharged the following day, with instructions to use traction nightly at home. He has had no further recurrence of pain.

CONCLUSIONS.

1. Headache and facial pain are common manifestations of cervical disc pathology.

2. When the typical pain is reproduced by head, cervical spine or nerve trunk pressure, pathology of the cervical discs is almost certain to be the cause.

3. Simple mechanical methods are usually sufficient to afford prompt relief of pain.

4. Surgical relief of nerve root compression is rarely necessary.

PRACTICAL TESTING FOR PURE PHYSIOLOGICAL HEARING FUNCTION.*

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The objective of this presentation is not to report newly discovered hearing tests. I am convinced that otologists are not suffering from a shortage of such tests. I will instead report to you what I found to be the best way of employing the long existing and well established hearing tests for estimating the correct degree and type of true hearing function still existing in the individually deafened ear.

I say "true hearing function" because there seems to be some confusion between the meaning of testing for the hearing function of a deafened ear and the testing for the ability of a deafened person to interpret correctly what little he hears with a deafened ear. I find it necessary to call attention to this distinction in the objective of testing hearing because I strongly feel that only the determination of true hearing function should be the concern of the otologist.

Only by determining the type, degree and extent of pure hearing function existing in a given ear can the otologist place himself in a position to correctly advise and institute when possible the necessary available means for improving physiological hearing in a functionally impaired ear.

In the presence of a hearing loss which is found by the otologist to be functionally incorrectible, the testing of a thus deafened person's ability to hear *ad libitum* with his limited

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hearing function, as well as the instituting of all available measures for improving his *ad lib* hearing should be left to the educator of the deafened.

I believe that the otologist's function in examining the hard of hearing patient should be to determine:

1. *What* each deafened ear fails functionally to hear under normal circumstances;
2. *Why* the individually deafened ear fails functionally to hear what it should normally hear under normal circumstances;
3. *Whether* the individually deafened ear which fails functionally to hear what it should normally hear under normal circumstances can be made to hear all or most of what it failed to hear, with the aid of abnormally amplified sound in one way or another;
4. *Whether* the ear which is not able to hear as it should normally under normal circumstances but can be made to hear more normally by abnormal amplification of sound is still physically in good enough condition so that it could lend itself to the improvement of hearing by time-proven available medical or surgical means, so that the use of amplified sound will not be necessary;
5. *Whether* the hearing loss is of progressive nature, making it mandatory for the otologist to stress the importance of instituting medical or surgical measures for the arrest of such progress whenever possible.

For too many years hearing was being tested mainly to determine whether or not a hearing loss existed. Since there was very little known about improving hearing, the importance of accurately determining the type of hearing loss in a given ear and measuring its exact degree and extent was, therefore, not so essential.

Today, some definitely time-proven ways and means of improving hearing in certain types of deafness already exist; also, there are promising investigations being conducted

which may lead to the ability of improving hearing in hitherto considered incurably deafened ears. In view of these facts, the necessity of carefully and accurately determining the type, degree and extent of an existing hearing function loss before advising measures for its relief and for determining the exact status of true hearing function after such measures have been instituted has risen to great importance.

I have been employing what I found to be the least fallible technique for testing hearing. It enables me to correctly diagnose the type, degree and extent of an existing loss of physiological hearing function within the deafened ear.

Both a correctly elicited history of the hearing impairment and a carefully conducted physical examination of both ears, prior to the testing of hearing function, are very essential.

The history of the onset and progress of the impaired hearing is preferably elicited not from the patient alone, but also from one closely related to and in constant association with the patient. To obtain a correct history of deafness from a patient seeking advice requires great clinical experience with the various middle ear, inner ear and mixed forms of deafness.

An understanding of the psychological factors, which will often prompt a deafened person to give a history of his deafness which can be very misleading to the less experienced otologist, is also essential.

After having taken the history I carefully examine the auricle, external auditory canal, tympanic membrane and middle ear structures and cavity of both ears with good illumination and if necessary with magnification. The findings are noted and an attempt is made to correlate the physical findings with the history given. If the physical findings are such as to render the veracity of the history doubtful, an attempt is made to obtain a more accurate history so that the physical findings and history could appear to be more plausibly correlated.

Having completed the history and physical examination, the testing for pure unadulterated physiological hearing function is begun in the following order:

1. Audiometry for pure tone sound.

2. Tuning Fork Testing for pure tone sound.

3. Speech Testing for ability to hear spoken words.

Audiometry for pure tone sound when executed patiently, carefully and objectively with a properly maintained correctly calibrated audiometer, the performance of which is constantly checked and verified, is today one of the most scientific means of measuring the degree and extent of an impairment of physiological hearing function for both air-conducted and bone-conducted pure-tone sound.

I sincerely believe that for the good of otology, no otologist who treats the deafened, either medically or surgically, should ever personally take audiometric readings either before or after having initiated such treatment. All audiometry is best performed by a well trained assistant whose sole interest should be to aid the otologist in making the correct diagnosis and to guide him in the correct evaluation of the results following his treatment. In my entire career I have never personally taken an audiometric reading either before or after instituting treatment for deafness. To date I do not know how to use an audiometer.

By carefully studying the decibel level of hearing for pure air-borne and bone-conducted sound in all the low and high frequencies, including the decibel differential between the air-conducted and bone-conducted sound, a correct diagnosis of the existing type of hearing loss in a given ear can be readily reached.

Tuning Fork Testing for pure tone sound, I employ with the following two objectives in mind: Since surgical treatment for deafness is indicated only for middle ear deafness and since it is to the patient's advantage to operate upon the ear with the greater impairment in air-conducted hearing, I first try to determine in which of the two ears the hearing

impairment for air-borne sound is worse. Immediately before striking, placing and firmly pressing the 512 frequency steel tuning fork against the median line of the skull or of the inferior maxilla with teeth tightly shut, I slowly, clearly and loudly tell the patient that I know as well as he knows in which of his two ears his hearing is better; however, what I would like him to do is to carefully observe and tell me in which of the two ears does he hear this particular tuning fork better, while disregarding the fact that the better of his two ears is known to him. By supplementing the Weber test with such careful explanation to the patient of what is expected of him to observe and report, and using the 512 fork instead of a lower frequency vibrating fork, I was able to make the following observations:

a. The 512 tuning fork is heard equally in both ears when the cochlear nerve function is about equally good in both ears and the hearing for the air-borne sound is equally good or equally bad in both ears.

b. The 512 tuning fork is equally not heard in both ears when the cochlear nerve function is equally bad in both ears and the hearing for air-borne sound is equal or not equal in both ears.

c. The 512 tuning fork is heard in one ear only when the cochlear nerve function in that ear is good and hardly any or no cochlear function exists in the other ear.

d. The 512 tuning fork is heard better in one ear when the cochlear nerve function is better in that ear, regardless whether the air-conduction hearing is equal or inequal in the two ears.

e. The 512 tuning fork is heard better in the ear with the poorer hearing for air-borne sound when the cochlear nerve function is equal in both ears.

My second objective of tuning fork testing for pure sound is primarily the determination of the approximate degree of unused good cochlear nerve function reserve still existing in

an air-conduction deafened ear, which could be utilized advantageously by a surgically reconstructed and functionally improved air-conduction mechanism.

For the above stated purpose the use of the 512, 1024 and 2048 frequency steel tuning forks is most valuable.

I always do these tuning fork tests myself. I believe that these tests should always be done personally by the otologist whose opinion and advice the deafened patient is seeking. This is also my opportunity to check the audiometric reading taken by my assistant. When my findings with the tuning fork tests are not in agreement with my assistant's audiometric reading, I order that the audiometric reading be taken again; however, in doing so I always insist that the recheck of an audiometric reading is done by an assistant other than the one who made the original audiogram, and without having had or being given the opportunity of looking at the audiogram which is being questioned. An assistant whose audiometric reading is being questioned, when permitted to retest the same patient, will as a rule not come back with greatly revised findings since he is not likely to admit having been careless in the first instance.

In testing with tuning forks the hearing of deafened persons who are seeking relief by vestibular fenestration, the important thing is to determine not how long they claim to have heard a particular tuning fork by air or bone conduction but rather to determine accurately the moment when they actually ceased to hear the tuning fork, which they claim to have heard. The determination of auditory acuity cannot be left entirely to the subjective response of the patient since this often leads to an erroneous estimation of the hearing status.

The veracity of the voluntary subjective responses to tuning fork testings for pure-tone sound, when these tests are executed in the traditionally accepted and employed manner, is often questionable. Especially so since many deafened persons with little hope for regaining their hearing in their desire to

be declared as suitable for surgical intervention will not hesitate to try their best to convince the examiner that they have good bone conduction hearing which they almost all know to be a "must" if such surgery is to be considered.

In the presence of this desperation psychology among many of the deafened, I have learned long ago not to rely entirely upon their voluntary subjective responses. I investigated and found involuntary emotional signs and involuntary objective responses which vary with the presence and absence of hearing. The following involuntary signs and responses when observed have been found to be infallible and, therefore, I consider them as indispensable to the testing of hearing with the steel tuning forks:

1. The delayed raising of their hand as indication of not hearing the pure-tone sound at all.

2. The spontaneous joyful grin and elated facial expression immediately upon contact of the temporal bone as an indication of hearing the sound.

3. The sudden change of the joyful grin to the sad, forlorn look as an indication of having suddenly lost the sound.

4. The deviation of the eyeballs in the direction of the formerly heard sound as the first indication of their search for the suddenly lost sound.

5. The turning of the head in the direction of the previously heard, but now lost, sound when failing to locate it by deviation of eyeballs.

6. The final shutting of their eyes to concentrate on finding the lost sound when everything else failed.

The obvious interpretation of these emotional changes of expression and the involuntary responses to tuning fork testing permits the correct determination and evaluation of their

hearing function for both air-borne and bone-conducted pure-tone sound as well as the correct time relationship between the two.

In order correctly to observe and evaluate these involuntary responses to sound when testing the hearing with the steel tuning forks, I invariably insist that the patient keep his eyes open and looks straight at me. I ask him to raise his hand as soon as he picks up and hears the sound of the tuning fork and drop his hand as soon as he begins to doubt whether he still hears the sound. By watching constantly for his involuntary responses to the sound and comparing and checking them with the voluntary responses, I find it possible by employing the involuntary responses as a control for the patient's voluntary responses to arrive at the correct estimation of the exact relationship between the air-conduction hearing and the hearing by bone conduction which can indicate whether non-utilized cochlear nerve function reserve is still present and when present its degree can thus be fairly accurately judged.

For testing with steel tuning forks, the 512, 1024 and 2048 speech frequency forks are used. After striking the tuning fork with a rubberized hammer, it is held close to the external auditory canal of the ear to be tested, while the other ear is being masked, until it becomes evident from both the voluntary subjective and the involuntary objective responses that the fork is no longer heard by air-conduction. Without striking it once again, the fork is quickly switched to the back of the auricle and placed forcibly and held tightly against the skin covering the mastoid process. Thus the ear is forced to pick up the pure tone sound by bone-conduction at an intensity which can no longer be heard by air-conduction. By carefully observing and correlating the patient's voluntary and involuntary responses to the bone-conducted sound, and timing the duration of its perception in a given air-conduction deafened ear, we can thus more or less correctly measure the degree of good cochlear nerve function reserve which is not being utilized by the functionally impeded air-conduction mechanism.

SPEECH-TESTING FOR DETERMINING WHETHER THE HEARING DEFECT IS CORRECTABLE OR NOT.

To determine whether dormant cochlear nerve function reserve which is not being utilized by a functionally impeded middle ear exists, the presently employed methods of testing hearing for the spoken voice are often misleading. These tests are inadequate because they are conducted in the language most familiar to the patient. The responses of the patient to words or sentences in the language best known to him do not necessarily represent pure cochlear nerve function alone. In many instances the responses to this type of speech-testing represent and are the result of a mixture of part cochlear nerve function and his ability to *ad libitum* interpret that part of the spoken word which the cochlear nerve function failed to supply. Such *ad-lib* hearing will vary with the I. Q. of the patient. This type of speech-testing belongs to the educator of the irreparably deafened patient.

Although, as a result of my great experience in diagnosis and treatment of deafness, I am thoroughly convinced that the degree to which the ability of an ear to hear the spoken voice has been reduced or improved can be readily judged from carefully taken audiometric readings of both air- and bone-conducted pure-tone sound, I, nevertheless, feel that in mixed forms of deafness, when the prognosis may be doubtful, speech-testing can often be a determining factor and govern the advice to be given the patient; however, if speech-testing is to serve best both the otologist and the patient, then such testing must be conducted in a language completely unknown to the patient or, still better, in meaningless words. The degree of a deafened person's ability or inability to repeat words with no meaning, when spoken into an individual ear with the other ear masked, is always representative of the degree and measurement of pure cochlear nerve function present in an ear. When employing speech-testing for diagnosis and advice, the degree to which such hearing is improved with the aid of the old-fashioned ear trumpet in a given ear represents the minimum amount of improvement that can be expected following the fenestration operation.

The following recorded meaningless words, or any other list of meaningless words, can be used for such testing:

boomsat	pirhum	askwig	browhard
speerisk	itchfitch	troopgrace	ceboo
booldrop	waycake	thudpuff	winkshank
spitbook	jesoon	icepass	chetnietz
hissteet	chicherin	cobhitch	svietlo
trapad	chetwierg	peepreap	poydyom
sismump	prosoutswo	shovetug	parachod
rumgup	baraban	chestboost	zavstrak
spissbice	snyeg	buckmast	
setdug	panavod	jaggap	
subnoy	zissquiz	gibzinc	

CONCLUSION.

Experience has taught me that by carefully conducted cooperative testing of the deafened with the participation, direction and supervision of an otologist with good clinical experience and judgment, the difference between a correctable and noncorrectable true physiological hearing defect is easily recognizable.

TRAUMATIC PARALYSIS OF THE VOCAL CORDS.*†

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Although traumatic injuries of the larynx represent a negligible percentage of all types of trauma, with current methods of high speed travel, more laryngeal injuries are seen now than formerly. Most of these occur when the larynx is struck on the steering wheel of an automobile, causing a crushing injury of the thyroid or cricoid cartilage. The trachea is less liable to injury than the cartilages of the larynx because of the cushion of the thyroid gland, but injuries to the trachea are also encountered. Laryngeal injuries do not, as a rule, produce paralysis of the recurrent nerve, but the distortion and scar resulting from laceration of the laryngeal mucosa cause distortions within the larynx which either produce fixation of the vocal cords or limit the motion to such an extent as to simulate paralysis of the cord.

One of the main complications of thyroid surgery is injury of the recurrent laryngeal nerve. The incidence of such injuries varies from 1.5 to 3 per cent of operations on the thyroid, and such accidents are ever present possibilities. Most cases of abductor paralysis are a result of thyroid surgery. Ever since the advent of thyroid surgery the correction of this complication has been a problem for the laryngologist.

The history as well as symptoms of abductor paralysis is typical in the majority of cases. Immediately following operation the patient is unable to speak except in a hoarse voice or whisper. Difficulty in respiration occurs only in patients in

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whom there has been much postoperative reaction and swelling or hematoma. In these cases tracheotomy is performed early. In the majority, however, the voice gradually improves, and by the time the patient leaves the hospital the voice is much stronger, although not normal. There is gradual improvement of the voice and with this gradual increase in respiratory difficulty. This becomes more severe with any form of physical exertion and upon lying down, when there develops a respiratory crow which is disturbing to other members of the household as well as to the patient. As this distress becomes more pronounced, activity becomes limited, and in order to obtain any rest the patient must sleep sitting up or propped up on several pillows. It is because of this increasing dyspnea that the patient soon seeks medical aid. The development of severe dyspnea is a gradual process requiring from six to eight months following injury.

If it has been necessary to perform a tracheotomy at the time of thyroid surgery or soon afterwards, it is advisable to wait six months before attempting restoration of the airway. This is to allow time for adjustment of thyroid patients, especially those with toxic disease. If, however, a tracheotomy has not been performed, then this should be done as soon as distress is noticed or when the patient is seen for the first time. This allows time for the patient to become adjusted to the tracheotomy tube and to overcome the immediate tracheitis and mucus associated with a recent tracheotomy. As soon as this period of adjustment has been accomplished, establishment of the airway may be attempted.

Since 1938, when Hoover[1] advocated submucous resection of the vocal cord in such cases, many surgical procedures have been suggested. Submucous resection provided an adequate airway in a high percentage of cases but resulted in a poor voice. Four years prior to Hoover's recommendation, Havens[2] had used a tracheotomy tube with a flap which enabled air to be exhaled through the cords but closed on inspiration. This provided a good speaking voice but made it necessary for the patient to wear a tracheotomy tube indefinitely.

It is, naturally, of primary importance to obtain an adequate airway without tracheotomy; a good voice, though desirable, is a secondary objective. To accomplish both, of course, would be ideal. With these objectives in mind, King,[3] in 1939, devised an operation in which the omohyoid muscle was used in an attempt to retract the cord for respiration and to establish some function of the cord for phonation. Kelly's[4] improvement of this technique consisted in arytenoidectomy via a window made through the thyroid cartilage. The latter procedure has resulted in a large number of successful results in his hands. Orton[5] modified Kelly's operation by removing the lateral half of the thyroid cartilage and then suturing the thyroarytenoideus muscle to the external perichondrium and the anterior split sternothyroid and thyrohyoid muscles. In 1946, Woodman[6] published his modification of the extralaryngeal approach; he advised a more open approach and a wider field for removal of the articular part of the arytenoid cartilage and fixation of the vocal cord laterally to the thyroid cartilage. Recently Thornell[7] proposed an intralaryngeal approach for removal of the arytenoid cartilage by means of suspension laryngoscopy. As this brief historical review shows, a variety of procedures and modifications of these has been attempted for relief of paralysis of the vocal cords.

I have had an opportunity to use several of the procedures mentioned in the preceding paragraphs. My experience with these will be briefly described. Like most other operations, these procedures have proved more successful in the hands of the originator than of those attempting to follow, until experience has made the pupil equal to the master.

The Kelly procedure, in which a window is made in the thyroid cartilage and the arytenoid cartilage is removed through this opening, was performed by me on three patients in 1945. The paralysis was the result of thyroidectomy for carcinoma of the thyroid in one patient; the result of the corrective operation was excellent in this case. The paralysis in the second case was produced by a traumatic injury to the larynx following a crushing blow to the thyroid cartilage;

the result of the Kelly operation in this patient was good. In the third patient the paralysis followed thyroidectomy for toxic goiter and the result was good, but the voice was poor, as both sides had to be operated upon in order to provide an adequate airway. The advantage of the Kelly procedure is that it enables the surgeon to grasp the cord at the vocal process and the pull is anterior and lateral, a fact which assures one of a more adequate airway. The second suture, placed at the middle of the cord, pulls directly laterally and this reinforces the first suture and enables one further to open the cord anteriorly, a feat which is difficult to perform by other techniques. The procedure has the disadvantage of requiring the surgeon to operate in an extremely small space. This makes dissection and removal of the arytenoid extremely difficult; furthermore, it is hard to maintain a dry field from bleeding muscles. These make the procedure tedious and rather long, but there is less reaction to the soft tissues of the larynx with resulting edema than in Woodman's procedure. I have since replaced Kelly's operation by the Woodman procedure.

The incision of the Woodman procedure is more extensive than that of the Kelly operation and gives a much wider field, better exposure and more adequate control of bleeding. I have observed that if the incision is made even more laterally than Woodman suggested, so that it is just at the anterior border of the sternocleidomastoid muscle, a more direct approach to the under surface of the thyroid cartilage is provided. One must be sure to disarticulate the cricothyroid articulation completely. Otherwise it is extremely difficult to rotate the thyroid cartilage for adequate exposure of the arytenoid. Also, more care has to be taken with this procedure to prevent tearing of the mucous membrane of the larynx. There is, moreover, a great deal more reaction locally and at the site of the arytenoid postoperatively than following the Kelly procedure; however, the advantage is that there is a great deal more space in which to work; the end-results have been satisfactory. Anterior lateral traction is obtained by hooking the suture around the lesser cornu and lateral traction by making

a drill hole in the thyroid cartilage. Of the 12* patients operated upon to date, good results were obtained in all but one case. In that case the cord was overcorrected, and although the airway is excellent the voice is poor, but the patient is perfectly satisfied to be without the tracheotomy tube after five or six years.

The Thornell intralaryngeal operation is relatively new; I have had experience in only four cases. The approach is much more direct than the extralaryngeal route, and the operating time is greatly reduced. Removal of the arytenoid is not difficult with adequate exposure and in young persons, especially women, a great advantage is the absence of an external scar; however, the end-results have not been so good as with the external approach in my hands. In two of the cases an adequate airway and a good voice were obtained. In the first case the poor result was due to too enthusiastic use of the diathermy, which resulted in cicatricial contraction of the interarytenoid space as well as the lateral area. This patient required plastic surgery with eventual skin grafting before an adequate airway was established. The final results have been extremely satisfactory with a good voice. In the fourth case the airway has not been sufficient for full activity.

It has been my experience that in the internal approach the voice is as good or better than in the external approach, but the airway is not so good; therefore, as the primary objective is to provide adequate breathing space and, secondarily, a good speaking voice, I prefer the external approach and specifically the Woodman technique.

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VASOMOTOR RHINITIS.*

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That there is still no unanimity of opinion regarding either the etiology or the treatment of vasomotor rhinitis is evidenced by the abundant literature. In the cumulative quarterly index for the years 1944 to 1948, there appear no fewer than 87 articles under the heading, "Vasomotor Rhinitis." From the majority of these one is left with the impressions that *a.* vasomotor rhinitis is due to a hypersensitivity to some foreign protein, be it inhalant or food; *b.* antihistaminics will relieve its symptoms and *c.* that specific desensitization or dust or histamine therapy will cure the condition. Experience, my experience at least, contradicts these impressions.

Vasomotor rhinitis should be thought of not as a disease of the nose but as a manifestation of a systemic disease, the result of which is a sympathetic-parasympathetic imbalance resulting in peripheral vasodilatation, edema and secretion.

There is ample evidence to support the concept that a sympathetic-parasympathetic imbalance is the mechanism causing vasomotor rhinitis. E. P. Fowler, Jr.,[1] reported a case of unilateral vasomotor rhinitis following the resection of a superior cervical ganglion. The results of blocking the sphenopalatine ganglion are well known clinically. I[2] reported some 90 cases in 1932. Experimental evidence is presented by Blier[3] in which he extirpated the sphenopalatine ganglion in dogs and obtained marked vasoconstriction in the nose on the side of the extirpated ganglion. He also showed that the

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parasympathetic fibres were the only ones with cell stations in the sphenopalatine ganglion. Corroborative evidence is afforded by the effects of the sympathomimetic drugs in the control of vasomotor rhinitis.

What then are the factors leading to such an imbalance? They can be stated as: *1.* Allergy. *2.* Endocrine dysfunction. *3.* Dietary inadequacy. *4.* Psychosomatic factors and a combination of any of these.

Allergy: I do not propose to elaborate on the allergic factors at this time except to say with the possible exception of true seasonal cases of allergic rhinitis, I find that allergy is not a common cause of the vasomotor rhinitis that we see repeatedly in the office. Bacterial allergy is perhaps one exception to this. There is considerable evidence that chronically infected membranes in the sinuses may give rise to an allergic rhinitis in which the bacteria in the membrane are themselves the responsible sensitizing proteins.

Endocrine Factors: I have neither the time nor the ability to discuss the mechanism of endocrine control of the autonomic nervous system. I would, however, like to mention cases illustrative of endocrine dysfunction resulting in vasomotor rhinitis.

Thyroid: Attention has been called to the part that thyroid plays in nasal disturbances by Proetz,[4] Laub[5] and others. I was first intrigued by this in 1932 when an Italian girl of about 20 years of age presented herself at the clinic for tonsillectomy. Her reason for wishing the operation was that her voice had become abnormal. It had assumed a nasal quality and was somewhat hoarse. She was an attractive girl who seemed, however, somewhat dull and apathetic. She had dry hair, dry skin and no vivacity. Examination showed her tonsils to be small and neither red nor obviously infected. She gave no history of sore throats. The chief finding on the examination was a typically "vasomotor" nose. The turbinates were purplish, boggy and swollen. There was some, though not excessive, secretion. Her mother, who accompanied her, stated that the girl was constantly tired, that she

would retire to bed at 8:00 P.M., and it was with great difficulty that she was aroused to go to work the following morning. She was sent for a basal metabolic test and the B.M.R. was reported as minus eight. In discussing this case with the endocrinologists, they assured me that we could, under no circumstances, consider this a hypothyroid patient. They advised strongly against the use of thyroid extract in this case. I did, however, put the girl on increasing doses of thyroid and after about five weeks, when she was taking three and a half grains a day, she came back for examination with what appeared to be a perfectly normal nose, and her mother at that time stated that she was an entirely changed individual. She no longer needed much sleep. She had many dates and was out at parties and dances constantly. Indeed the girl had changed considerably in her personality. In the place of a rather phlegmatic, lifeless looking individual, there appeared a girl very bright and full of the joy of living. Another similar interesting case presented in a boy, M. B., 12 years of age, who was brought to me because, as his mother stated, "he has a cold all the time." This boy was fat, pale and without much vivacity. His mother told me that after school he would come home and sit and read a book and that he never wanted to go out and play with the other boys. His nose presented the typical appearance of vasomotor rhinitis, the rather pale, boggy turbinates with a fair amount, in this case, of clear, watery mucus. His basal metabolic rate was minus 19. We started him on thyroid extract and gradually increased the dose. At one time he was taking as much as 3½ gr. a day. His basal metabolic rate under this regime remained at minus 20. He was carried on with thyroid therapy, 2 gr., and for the past five years has had no colds nor any respiratory difficulty. After he had been on thyroid for awhile, his mother told me that he was a changed individual, that he now was full of energy, played with the other boys and indeed had been arrested on Halloween for some mischief which the gang had gotten into. He had, in other words, become a real boy.

We followed many of these cases of vasomotor rhinitis with low basal metabolic rates, and we have found that where a

low basal metabolic rate is combined with a high blood cholesterol, the results from thyroid therapy are good. With a low metabolic rate and a normal blood cholesterol there are fewer good results. The least successful cases for thyroid therapy are those with a near normal basal metabolic rate and a normal blood cholesterol.

It is important in these cases to give sufficient thyroid to be of value. Our usual method of giving it is to start the patient with 1 gr. a day for perhaps two weeks and then increase to 2 gr. for two weeks to 3 gr. a day for two weeks and so on, being guided in the increase of the dose by the appearance of the nose. As a rule, about 3 to 4 gr. are necessary to cause a change in the nasal mucosa. When the desired change has been accomplished, we then reduce the dose until we find the optimum maintenance dose for the particular patient.

Ovarian Dysfunction: The relation of the nasal mucosa to ovarian function is well known and is evidenced by the changes observed during menstruation and pregnancy. We are all familiar with the occasional nasal blockage which occurs in the latter part of pregnancy which is immediately relieved when parturition has been accomplished. These same changes may occur as a chronic vasomotor rhinitis in some women at the menopause or in others who have been castrated. As an example, I would quote the case of Mrs. E. B. This woman of some 45 years of age was seen in 1941 with a bilateral chronic maxillary sinusitis, nasal polyps and much mucopus in both sides of the nose. She was diagnosed as having chronic maxillary sinusitis and had a bilateral Caldwell-Luc operation. Following the operation, when the nose had healed, the antrums could be washed and no pus obtained. The patient, however, constantly complained of a cold and when she was examined presented a red, rather dry and definitely swollen membrane. This is the type that Proetz[4] has described in his paper, "The Thyroid and the Nose." I was misled by the appearance and thought that this woman did have repeated respiratory infections. She was treated for these with various local treatments and with vaccine but

with no success. After considerable time, in 1944, I was talking to her one day and trying to get some line on any general condition which might be contributary to her nasal state, and when I asked about her menstrual periods, she informed me that some years before she had had her uterus, tubes and ovaries removed and had not menstruated since. She was started on Diovocylin by injection once a week, and in three weeks her nasal condition cleared completely; she was followed thereafter by sublingual medication with Estrogen and for the next three years was seen at regular intervals without any complaints. She had no further respiratory infections and no further nasal trouble.

Testicular Hormone: Just as the nose reacts to abnormal function of the ovaries, so it reacts to abnormalities of testicular function. This has not been as publicized nor as widely observed as the condition in the female. Laub[5] reports a case of vasomotor rhinitis and polyps in a patient at the male climacteric. I would like to quote a case, I. B., a medical student, 24 years of age. This boy as a senior medical student came to the nose and throat service to do an elective. He had just recently been married and one day came to me complaining that he was deaf in both ears and had what he described as a very bad cold. The nose was completely blocked with swollen, wet membrane. There was much clear discharge. Both ears were completely full of fluid and the diagnosis was upper respiratory infection with serous otitis. The eardrums were opened and the fluid obtained, and on smears, this fluid was found to be loaded with eosinophiles. The diagnosis clearly was "allergy of the upper respiratory tract and ears." He was referred to the allergists who did the usual skin tests, finding no positives. Thinking, therefore, that he might be sensitive to something about his newly acquired wife, we hospitalized him and kept him isolated from his wife. Incidentally, this caused quite an upheaval in the family. After a month of this isolation, his condition was unchanged and he was no better. Under the general premise of his recent marriage and its attendant duties, I put him on testosterone therapy. Within six weeks, his ears had completely cleared, his hearing was normal and his nasal mucosa presented a normal

appearance. He remained on testosterone therapy for some four or five months and since has been completely free of symptoms. I have known this boy well. He was in the Army and came back to specialize in otolaryngology and I am proud to have had him as a student.

Psychosomatic Factors: It is generally realized that emotional states may lead to peripheral vascular change. For example, the blushing that occurs with shame or embarrassment, the blanching that occurs with fear. I wonder, however, if otolaryngologists in general realize that emotional factors may play a large part in chronic organic changes, particularly in the nose. The experimental work which has recently been done at Cornell Medical School in this subject is particularly interesting. Work done in the department under the direction of Dr. George H. Wolff is well worth our attention. For example, in a paper entitled "Physiologic Mechanisms of Psychosomatic Phenomena," the authors indicate that changes may occur in the nasal mucosa and in the cellular response in the nasal mucosa from a psychiatric interview. They quote the case of one individual who suffered from what they termed chronic rhinitis. "A biopsy was taken from this person when he was in a period of complete rest and relaxation when membranes were in an average state of activity and again from the opposite turbinate at the height of a frustrating interview when the patient was on the verge of tears. Both biopsies were made with the same technique and the same topical cocaine anesthesia. The first section showed an essentially normal mucosal structure with moderate round celled infiltration. The second revealed the mucous glands to be filled with secretion and the vascular and lymphatic channels to be prominent and dilated. The cellular reaction of the nasal tissues was also greatly modified by symbolic stimuli. When the nasal secretions from the same subject were collected and stained by an appropriately standardized technique before, during, and after the discussion of significant conflicts, it was found that nasal hyperfunction was accompanied by marked eosinophilia, not only locally but in the peripheral blood as well." Consideration of this finding is extremely important to us when we have been told and we

have believed in the past that in the presence of nasal eosinophilia, allergy,—and I refer now to a protein hypersensitivity,—was the sole cause. We should, I believe, revise our concept in this regard. Further experiments by this group of workers have demonstrated that, for example, when a known hay fever patient was put into a room in which there was a known pollen count, frequently there were no aggravated symptoms. A little sniffling, a little rhinorrhea, but no change in the color or swelling of the nasal mucosa. This same individual when situations about his personal life and interpersonal relationships were discussed had marked symptoms of hay fever with lacrimation and so on, until again at the end of the interview he was reassured and the nasal symptoms subsided.

DISCUSSION.

That the patient suffering from a chronically stuffy nose, postnasal drip, frequent colds, "sinus" and so on is, on the whole, unsatisfactorily handled is due, I believe, to the unfortunate misconception regarding the etiology of the condition. Such misconceptions are due, in large part, to the nomenclature of the disease. "Allergic" is the usual term applied. If by "Allergy" we mean "altered reaction," it is an admirable term. Common usage of the term "Allergy," however, has come to connote an antigen-antibody reaction, or a true protein hypersensitivity. This is, I think, unfortunate because immediately all attention is directed toward discovering the offending allergen and if this is not found, a nonspecific, desensitizing regime with dust extract or histamine is instituted. The other important factors I have mentioned above are consequently overlooked. For this reason, I think "Vasomotor Rhinitis" is a better term. By its use we may be led to think of other factors than allergy which influence the vasomotor function of the individual.

Again I would quarrel with the too easy acceptance of eosinophilia as a diagnostic finding in allergy, to the exclusion of other possibilities. The reports of Wolff and his co-workers are certainly worthy of further consideration. Eosinophiles

are present in secretions and tissues in allergic rhinitis but their presence is not necessarily diagnostic of Allergy. They may be present in other conditions.

The fact that other causes of vasomotor imbalance, endocrine, dietary, psychosomatic, and so on are often overlooked may be due, perhaps, to specialism. A specialist must be constantly on guard against concentrating his attention on his particular part of the body at the expense of the patient as a whole. The otolaryngologist faced with a patient with vasomotor rhinitis is too apt to regard him as just a nasal or a sinus problem and forget that the patient has glands, a gastrointestinal tract, a brain, and for want of a better term, a soul. By these remarks, I do not mean to infer that we as otolaryngologists must also be endocrinologists or psychiatrists. I do insist that we should be able to recognize disease in other systems as causing symptoms referable to the ear, nose or throat and consequently seek the help of the appropriate specialist for further care. In these days it becomes a matter of team play between the otolaryngologists and other specialists if the patient is properly to be cared for.

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SURGICAL REMOVAL OF CONGENITAL NECK FISTULA.*†

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Any discussion of congenital cysts and fistulae of the neck immediately involves two somewhat controversial problems: The first, etiology and classification; second, the rôle of the laryngologist.

Such efforts as the author has exerted toward the interpretation of branchiogenesis have ended in utter confusion and bewilderment. This has resulted, no doubt, from a combination of mental apathy toward the seeming fantasy of embryology and the impatience of an average clinician.

Dr. Charles Blassingame,[1] in 1947, presented a paper to the American Laryngological, Rhinological ond Otological Society, Inc., which stimulated a renewed interest in the subject.

While several etiologic theories regarding the origin of congenital lesions have been proposed, none has been universally accepted. "It seems more likely," according to Ward[2] in a recent publication, "that all the various theories have an element of truth in them." Meyer's interpretation of Wenglowski's work[3] gives us a clearer conception of the etiology and a more orderly classification of congenital pathology in the neck.

The terms branchial and branchiogenic have been used so generally and loosely that their true connotation is often disregarded.

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Meyer arrived at certain conclusions which, if accepted, will help to organize our reasoning in this regard. These are (see Fig. 1):

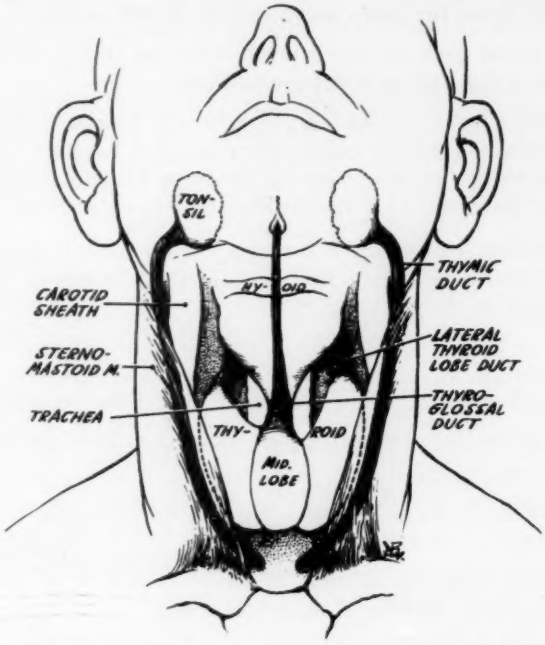

Fig. 1. Embryologic model projected upon adult contour for easier orientation (adapted from a model of Wenglowski).

1. The branchial apparatus disappears early in the second embryonic month. Only squamous rests, sometimes cartilaginous, can remain behind as parts of it. These are above and dorsal to the hyoid bone. Below the hyoid no rest of the branchial apparatus can remain behind. (Bailey and others have disputed this.)

2. The thymus develops from the third pharyngeal pouch. The thymic canal runs obliquely from the lateral pharyngeal wall to the sternum where the thymus gland develops.

3. The thymic canal retrogresses. It may persist throughout life or segments of it may persist.

4. The thymic canal rests may form a fistula or cyst. If the canal persists a complete fistula will result.

5. Lateral neck fistulae coincide with the thymic canal in direction and in histologic findings.

6. The lateral thyroid lobes have a short canal that disappears early in embryonic life. If a fistula or cyst resulted, the inner opening would be near the opening of the glottis. (If such exist, they are rare.)

He states definitely, "All congenital anomalies caused by the incomplete retrogression of the branchial apparatus must be located in the region above the lower border of the hyoid bone."

From these classifications we can make the following clinical classification of congenital cysts and fistulae (see Fig. 1).

1. Lateral cysts and fistulae which arise from the thymic tract.

2. Midline cysts and fistulae which arise from the thyroglossal tract and its ramifications.

3. Other cysts and fistulae which, if located at or above the hyoid, may be of branchial origin.

It would be presumptive, indeed, for the author to vouch for the academic accuracy of any embryologic research; however, from a clinical standpoint, the work of Meyer and Wenglowski gives us a concise classification and places the branchiogenic lesions in a limited anatomic area.

The treatment of these lesions is successful surgery. Successful surgery requires a knowledge of anatomy and entails the complete extirpation of the cyst or fistula.

The first case to be discussed was a complete lateral fistula which was totally removed with an excellent cosmetic result. This 19-year-old white boy presented a small fistulous opening about one inch above the right sternoclavicular articulation.

This had been present since birth and had intermittently discharged a sticky mucus. In recent years the frequency of this discharge had become a handicap to the patient. Lipiodol was instilled into the opening and radiographic studies revealed a tract upward to the pharyngeal region (see Fig. 2).

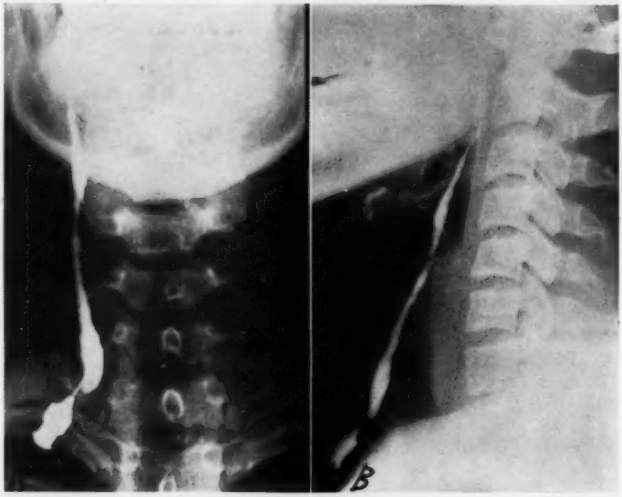

Fig. 2. Radiographic study of complete lateral fistula. (A) The tract follows the course of thymic duct.

Two weeks following a preliminary tonsillectomy the fistulous tract was removed surgically. A small filiform bougie which was passed through the tract greatly facilitated our surgical dissection. For cosmetic reasons the so-called "step-ladder" method was employed (see Fig. 3). Three transverse incisions were made, the first at the level of the fistulous opening. As the tract was dissected it was threaded from one incision upward to the next. Finally, when the thin wall of the pharynx was palpated, the tract was ligated, cut and removed from the neck wound (see Fig. 3). The three incisions were then closed. A very satisfactory result was obtained.

As Bailey[4] stated, "This step-ladder method leaves quite inconspicuous scars and robs dissection of a branchial fistula of most of its terrors" (see Fig. 3).

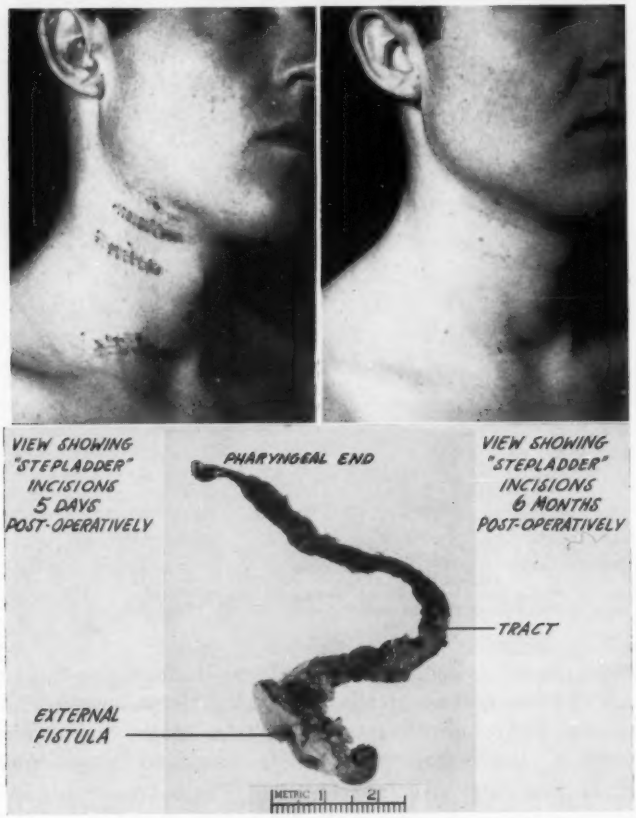

Fig. 3. Photos showing excellent cosmetic result. The fistulous tract appears smaller than at surgery due to fixative agent used prior to the photograph.

Radiographically and anatomically, this tract followed precisely the same course as that of the thymic tract (see Figs. 1 and 2). From the tonsillar fossa it extended laterally and

downward beneath the posterior belly of the digastric muscle. From here it passed medially and downward, anterior to the carotid sheath, to its opening along the anterior border of the sternomastoid muscle. This course of the thymic tract is said to be consistent. In one case we thought that an exception had been found because the opening was posterior to the sternomastoid, but the constant course of the thymic corridor was later proven when the tract revealed the fungi of actinomycosis.

The second case was a lateral cyst which was removed surgically. This 37-year-old white woman presented a large cystic mass in the right side of the neck just below the angle of the jaw (see Fig. 4). This mass had been present for eight

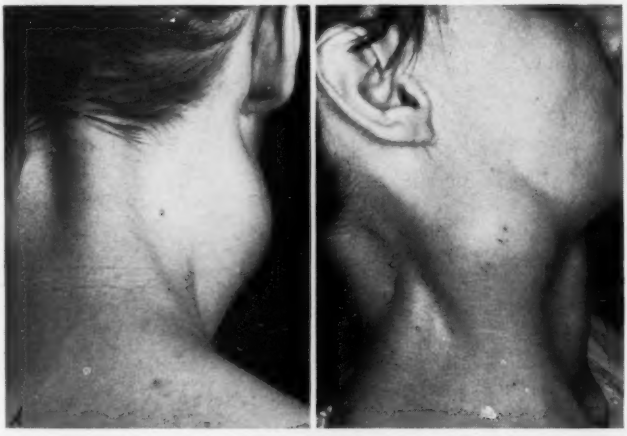

Fig. 4. Posterior and lateral views of lateral cyst. Small dark area in the lateral view resulted from the aspirating needle.

months. One ounce of opaque fluid was aspirated and lipiodol was instilled for radiographic study (see Fig. 5). The cyst was removed intact through a transverse incision. There was no duct or tract attached to it (see Fig. 6). It lay beneath the cervical fascia and upon the carotid sheath.

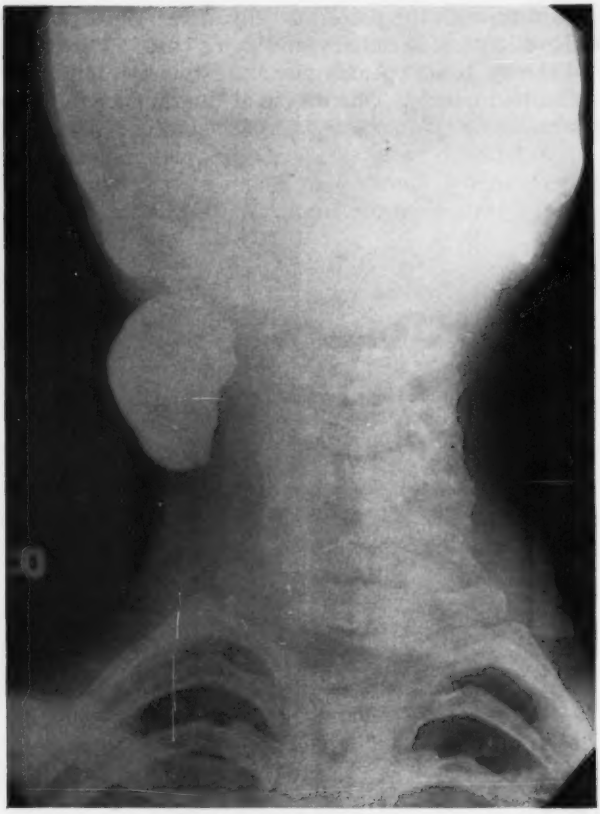

Fig. 5. Radiopaque study of lateral cyst.

As to whether this second congenital lesion should be considered of branchial origin seems academic. The important point is that it was characteristic of congenital cysts in this area. It appeared in adult life, was situated in the upper one-third of the neck and was filled with opaque fluid.

The question has been asked, "Are these lesions of the neck within the field of the laryngologist?" They are most assur-

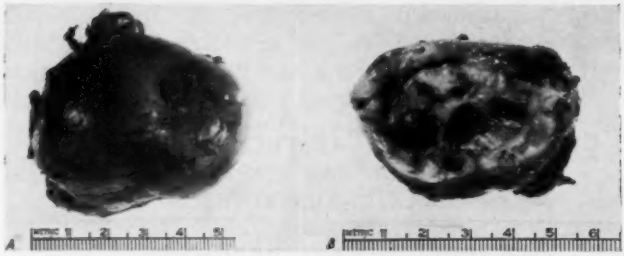

Fig. 6. Photos of specimen taken immediately postoperatively. (A) Intact specimen. (B) Inside of specimen after fluid was removed.

edly within the realm of the well-trained laryngologist. We should increase our efforts in the training of residents in this regard.

Certainly the pharyngeal end of a lateral fistula is in the field of laryngology, located as it is in the tonsillar area. With this pharyngeal attachment of lateral and thyroglossal tracts already in our favor, our interest and teaching should be such as to retain all congenital neck lesions in the field of laryngology. This can be attained only by the thorough training of residents. The diagnosis and surgical anatomy of congenital cysts and fistulae should be included in the programs of all teaching residencies in otolaryngology.

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CYTOLOGICAL SMEAR TECHNIQUE IN THE DIAGNOSIS OF CARCINOMA OF THE MAXILLARY SINUS:

PRELIMINARY REPORT.*

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and C. H. LUPTON, JR., M.D.‡,
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The cytologic method of cancer diagnosis has been attracting increasing interest since the publication of the monograph by Papanicolaou and Traut[1] in 1943. Papanicolaou and his associates in the study of exfoliative cells of the female genital tract developed a technique in the preparation and staining of vaginal smears so that neoplastic cells could be recognized. From these initial studies there have evolved methods for examining the cellular elements of the secretions and contents of other body areas,[2] such as the lung,[3] stomach,[4] urinary tract[5] and nasopharynx.[6]

We have applied the cytologic smear technique to the study of washings from the antra in an effort to evaluate its usefulness in making the difficult early diagnosis of carcinoma of this sinus. All too often the diagnosis of antral malignancy is not made until gross clinical and radiologic evidence of such involvement is manifest. Should an acceptable degree of certainty be demonstrated by the smear technique, then the relatively simple procedure of antral lavage could be more fully utilized as a diagnostic aid.

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The study of the cells derived from antral washings requires that the cytologist be familiar with the cells from noncancerous disease of the antrum such as suppurative, allergic, or other varieties of sinusitis.

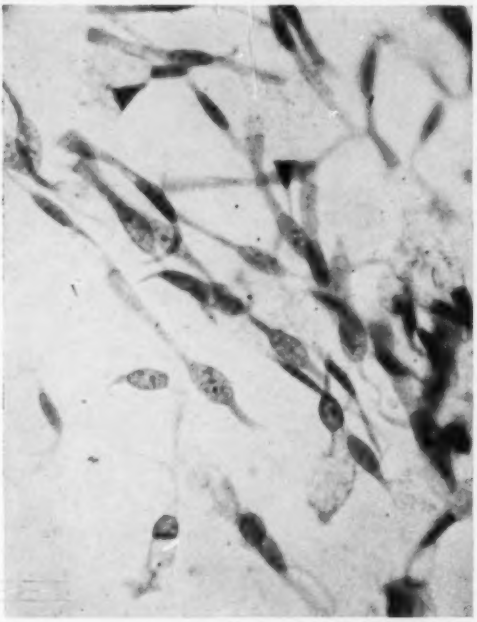

Fig. 1. Antral washings showing normal ciliated columnar epithelium. 690×.

In this preliminary report we have limited our cases to those in which malignancy could with reason be suspected; *viz.*, those presenting evidence of disease in a single antrum. Washings from 72 patients were studied, among which six cases of carcinoma were found. Unfortunately, all six of these cases exhibited cancer in advanced form which could have been recognized readily by other methods of diagnosis; however, since we were able to distinguish the tumor cells so readily in antral washings from these six cases, we are hopeful that earlier forms of the disease can be recognized with an equal degree of accuracy.

Technique: The majority of the antral irrigations were performed under topical cocaine or pontocaine anesthesia. Several lavages in children were done while under ether anesthesia. The antra were entered with a curved, blunt cannula

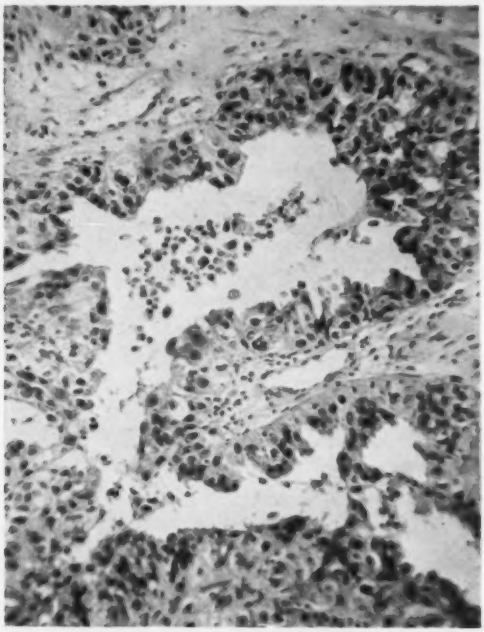

Fig. 2. Carcinoma of antrum showing exfoliation of tumor cells. 150×.

through the middle meatus, or with a straight needle through the inferior meatus, depending upon which approach was thought to be most desirable in the individual case. Warm normal saline was used as the irrigating solution. Approximately 50 cc. of the washings were collected and to this an equal quantity of 95 per cent ethyl alcohol was added. The combined solutions were centrifuged as soon as possible for 30 minutes at medium speed (3000 r.p.m.). The sediment was collected and spread evenly on slides previously coated with egg albumin. The slides were then immersed in a solution of

equal parts of 95 per cent ethyl alcohol and ethyl ether. The staining technique followed essentially that devised by Papanicolaou,[1] using hematoxylin, orange G-6 and E-A 65. The completed slides were mounted with cover slips and Canada balsam or permount.

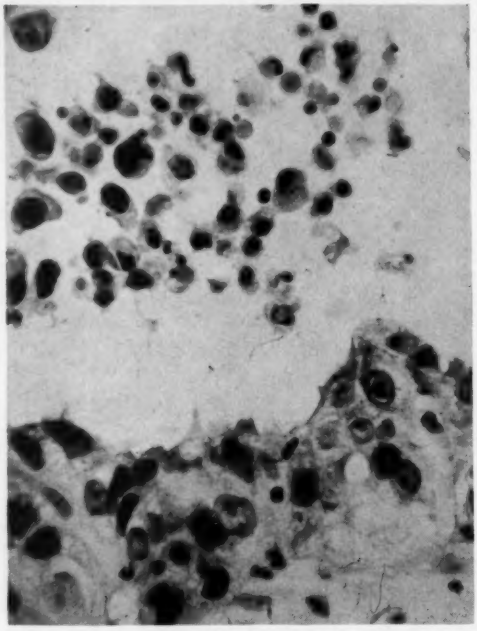

Fig. 3. Higher power magnification of Fig. 2. 550×. Note resemblance of exfoliated cells to malignant cells in tissue.

Microscopic Findings in Noncancerous Antral Disease: The washings from 66 patients were examined. As one would expect, the smears of the washings encountered in this group of patients contained a wide variety of cells. A few erythrocytes were usually present and easily recognized. As all the patients had some type of sinusitis, leucocytes were nearly always present. The polymorphonuclear leucocytes and lymphocytes were readily identified. Eosinophiles and mononu-

clear phagocytes were sometimes seen. A few stratified squamous epithelial cells may be seen, but most of the epithelial cells were tall, columnar and ciliated in type. Numerous variations may be encountered, such as columnar cells with no cilia, as well as oval and round cells, the latter cells being

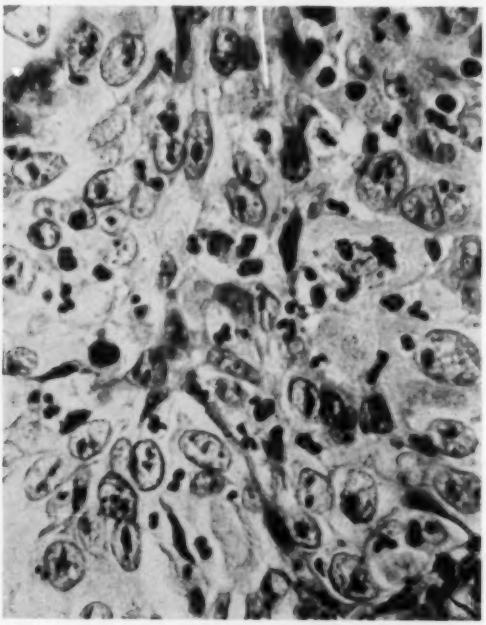

Fig. 4. Section of carcinoma of antrum (Case 1). 650×.

found most frequently with chronic suppurative sinusitis. The epithelial cells may appear singly or in small groups. Not infrequently degenerative cells were seen which could not be definitely classified. These may be epithelial or inflammatory in origin and at times may offer some confusion in diagnosis. Some of the cells encountered are demonstrated in the photomicrographs.

In reporting the findings from the washings the following classification as stated by Papanicolaou was used:

Class I: Smears that are negative, containing no atypical cells that may be confused with cancer cells.

Class II: Smears that are essentially negative but which contain atypical cells not generally confused with cancer cells.

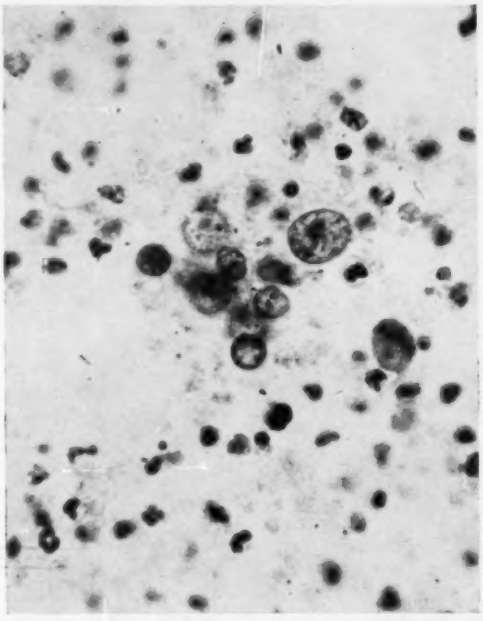

Fig. 5. Antral washings from Case 1 showing exfoliated tumor and inflammatory cells. 690×.

Class III: Equivocal smears, containing abnormal cells suggesting a cancer but insufficient in quantity and characteristics to justify a positive diagnosis of cancer.

Class IV: Smears containing individual cells and groups of cells with sufficient characteristics to identify them as cancer cells.

Class V: Smears similar to those in Class IV showing characteristics even more diagnostic of malignancy. In this

series no diagnosis of Class V was made, due to lack of experience in interpreting these washings.

Microcopic Findings in Neoplastic Antral Disease: Over a period of 12 months we have had six patients with carcinoma

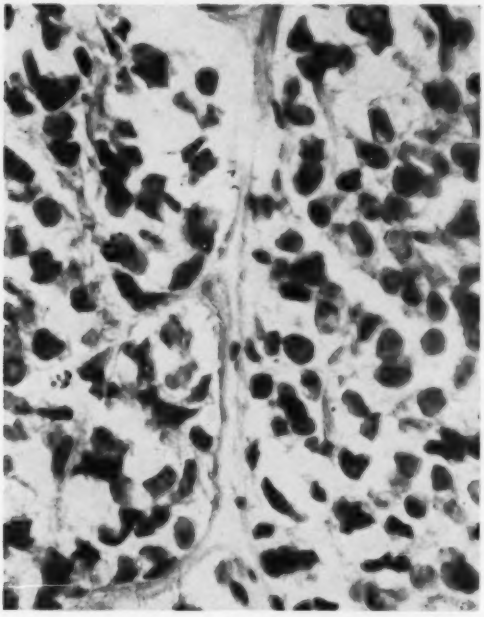

Fig. 6. Section of carcinoma of antrum (Case 5). 650×.

primarily of the antrum. There have been carcinomas of the nose, involving the antra secondarily, which have not been included in this group. The smears in the washings from these positive cases showed the same cellular elements as in the noncancerous group, with cancer cells in addition. Some of the cells are demonstrated in the accompanying photomicrographs. The cancer cells appear individually and in groups, with a fairly marked difference in the shape and size of the individual cells. The nuclei are large and abnormal,

with prominent nucleoli and abnormal chromatin arrangement within them. Basophilic staining reactions in these nuclei are frequently encountered. Mitosis is not often seen. In the epidermoid types the cytoplasm may show varying

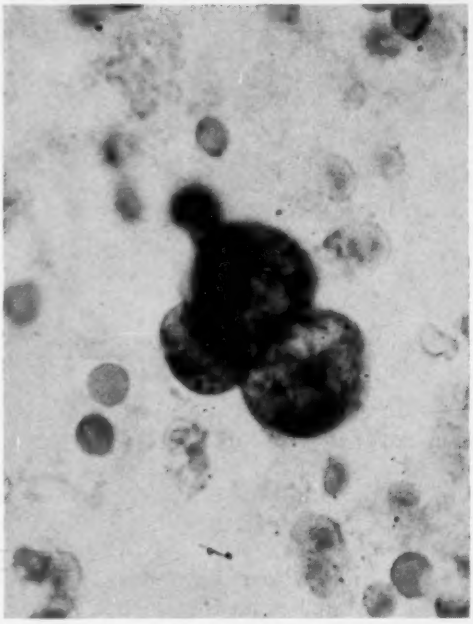

Fig. 7. Antral washings from Case 5 showing exfoliated tumor cells. 2100×.

degrees of keratinization manifested by orange color. The cytoplasm was unusually scanty when compared with that of the normal cell and may show vacuolization. It was mainly the abnormal characteristics of the nuclei of the suspicious cell or group of cells that led to a positive diagnosis of cancer.

Some details of these six cases, all proven by biopsy to have carcinoma of the antrum, may be seen in the accompanying table:

CLASS IV AND CLASS III PAPANICOLAOU STAINED SMEARS

	Patient	Sex	Age	Color	Class	Pathology Reports
1.	C.M.	F	42	W	IV	Epidermoid Carcinoma, grade II.
2.	H.C.	M	17	W	IV	Unclassified Malignant Tumor (Favor Endothelioma).
3.	H.J.	M	7	W	IV	Diffuse Endothelioma.
4.	J.G.	M	62	W	IV	Epidermoid Carcinoma and Adenocarcinoma, grade IV.
5.	D.H.	M	50	W	IV	Adenocarcinoma, grade III.
1.	C.P	F	21	C	III	Epidermoid Carcinoma, grade IV.
2.	V.M.	M	25	W	III	Chronic Maxillary Sinusitis.

Two Class III cases were encountered. One was shown eventually by biopsy to have carcinoma of the antrum and the other chronic sinusitis. To place these cases in a false negative or false positive category is open to some question, as Class III is a recognized equivocal group. Exploration of the antrum in patient No. 1 of the Class III series disclosed a carcinoma with intact and grossly normal mucous membrane over the tumor area (subsequent washings following preparation of this paper were reported as Class IV). Patient No. 2 in the Class III group was found to have chronic suppurative sinusitis upon exploration. This equivocal report was due most likely to cells exfoliated from an inflamed mucous membrane which had areas of metaplasia in its epithelium. It would appear that all Class III washings should be repeated in an effort to obtain a more satisfactory diagnosis.

Patient No. 4, after frequent washings over a six months' period, subsequent to X-ray therapy, became a Class II, then again returned to a Class IV with clinical evidence of recur-

rence of the carcinoma and positive tissue examination. This suggests that the Papanicolaou stained washings may be of aid in the follow-up of treated cases.

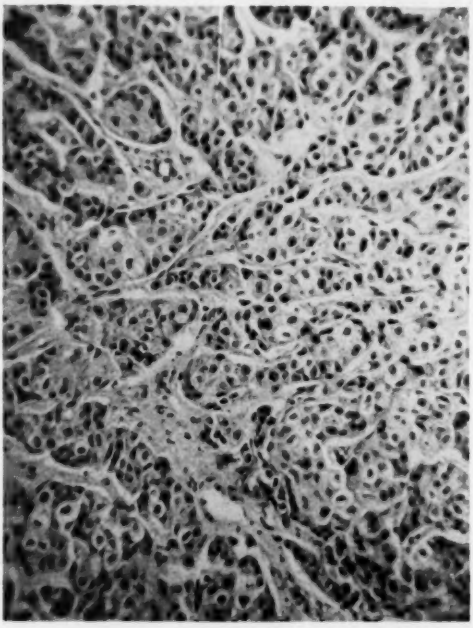

Fig. 8. Section of carcinoma of antrum (Case 4). 160×.

Patient No. 5 was interesting in that an initial Class IV smear became a Class I, following surgery and X-ray irradiation, and has remained so for over eight months. Tissue examination at that time was negative for recurrent carcinoma.

Another patient not included in this group with radiologic and clinical evidence highly suggestive of carcinoma of the antrum showed negative washings. Exploration disclosed a large dermoid cyst.

Comment: Among 72 consecutive cases of unilateral antral disease, six cases of carcinoma have been recognized without serious error by the application of the Papanicolaou technique to antral washings. The diagnosis of either sinusitis or carcinoma was later confirmed by the clinical behavior of the case or by biopsy.

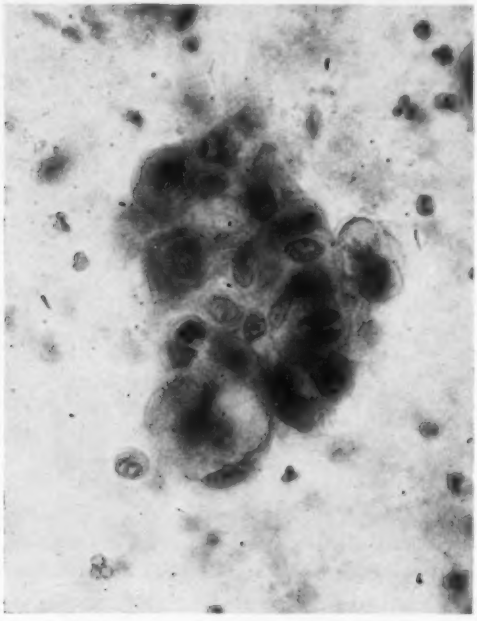

Fig. 9. Antral washings from Case 4 showing typical group of exfoliated cells. 690×.

It, therefore, appears that cytological studies of antral washings should be recognized as a useful aid in the diagnosis of carcinoma of this region.

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NEURINOMA OF THE PHARYNX. REPORT OF A CASE.

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This type of tumor is known by many names. Neuroma, neurinoma, neurilemmoma, neurilemnoma, solitary neurofibroma, gliofibrosarcoma, Schwannoma, peripheral glioma and fibroma of nerves can be considered synonyms. These tumors originate from the fibres of peripheral nerves. The exact origin is probably the neurilemma or Schwann's sheath of nerve fibres. These tumors are characteristiaclly ovoid in shape, well encapsulated and firm. The cut surface presents a glistening, gelatinous appearance and is quite firm. Histologically, there are spindle shaped cells which have a tendency to arrange themselves in parallel formation, the so-called palisading effect. The nuclei are also spindle shaped, containing nuclear chromatin and reticulum. There are usually one or more nucleoli. Interspersed between cells and groups of cells are reticulum fibres and sometimes a considerable amount of collagen. It is thought by some investigators that the occurrence of a tumor of this type is but a solitary manifestation of Von Recklinghausen's disease. Indeed, Von Recklinghausen's disease has been observed in several patients having neurinomas of the head and neck.[1]

Figi, in 1933, reported such a tumor in the pharynx, and said that he could not find a similar one in the literature. Since that date, about 15 such tumors have been reported as occurring in the pharynx. Undoubtedly there are many unreported ones. These neoplasms are always benign, slow growing and never cause trouble until they are of such proportions that their size produces symptoms. The most common symp-

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toms are difficulty in swallowing and altered speech. On examination the tumor mass is easily seen projecting into the pharynx. In some cases there has been noted a Horner's syndrome on the same side.[2,3] Diagnosis is usually established by pathological report of tissue removed at the time of operation. In some instances aspiration biopsy has been successfully undertaken prior to operation. The treatment is surgical, and there are no reported recurrences. Some surgeons advocate the external route[6,7] with the preliminary precaution of ligating the external carotid. Those men reporting the intraoral approach experienced no difficulty in accomplishing complete removal of the tumor mass without excessive bleeding. Postoperative complications have been Horner's syndrome, dysphagia due to paralysis of the soft palate, unilateral laryngeal paralysis. One early postoperative death was reported.[9] This followed carotid ligation and the exact cause of death was undetermined. The paralyses observed postoperatively are to be expected when one removes a tumor having its origin in nerve tissue. These neoplasms are extremely radioresistant, and Roentgen therapy is never advocated.

CASE REPORT.

In September, 1949, a somewhat obese, 25-year-old, white, married female was seen for the chief complaint of difficulty in swallowing, and impaired speech. Her mother had noticed enlargement of the left tonsil in her daughter since the age of eight or 10. For the past five years her voice had been affected and had an adenoidal quality.

Examination revealed a large mass in the left pharyngeal wall which displaced the faucial tonsil medially as far as the midline. The appearance was in many respects similar to a large peritonsillar abscess, without the signs of inflammation, of course. Palpation revealed a firm, but not overly hard, mass. There was no palpable pulsation. The remainder of the examination of the ears, nose and throat revealed nothing abnormal. Under general anesthesia with ether the tonsil was peeled off the tumor mass with ease, revealing the tumor to be behind the pharyngeal musculature. The muscle layer was split and an attempt was made to remove the tumor in one piece. It was a well encapsulated, ovoid mass of large proportions and was so firmly attached posteriorly that I could not deliver it in one piece. It was removed piecemeal with a tonsil snare, care being taken to avoid the external carotid artery which was found to be displaced laterally by the mass. The tumor was easily

removed in this manner, leaving a large cavity in the neck. Into this vacancy a finger could be passed superiorly to touch the transverse process of a vertebra high above the level of the soft palate. Inferiorly, the cavity extended well below the base of the tongue and the fingertip could be placed directly on the exposed and laterally displaced carotid sheath. There was minimal bleeding throughout the operation. Any approximation of the incised tissues was decided against, because of the possibility of inhibiting drainage, and the cavity was left open. Penicillin was given postoperatively, and convalescence was uneventful. Postoperatively, the patient experienced difficulty in swallowing and this has persisted to date. There is almost complete paralysis of the soft palate on the operated side. She manages solid food very well, but liquids have a tendency to return through the nasal passages. Her voice is normal.

PATHOLOGICAL REPORT.

Gross Pathology: The specimen consists of numerous varied sized masses of tumor tissue weighing 40 gm. and small amounts of attached fat and muscle. The tumor tissue is of a uniform moderately firm consistency and pale yellow in color.

Microscopic Pathology: The predominant histologic structure of the tumor consists of oval and fusiform nuclei lying in a palisade arrangement about rounded pink-staining areas free of nuclei. In smaller areas the tumor appears more collagenous without regimentation of cells. Mitoses are absent. This is considered to be a neurilemoma, type A.

SUMMARY.

1. Neurinomas are benign tumors having their origin in the fibres of peripheral nerves.

2. These tumors cause symptoms only by virtue of the size they sometimes attain.

3. Treatment is surgical removal; postoperative complications are paralyses of the nerves involved.

4. A case of neurinoma of the pharynx was presented.

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706 Church Street.

AN UNUSUALLY LARGE TRACHEAL FOREIGN BODY (FOUNTAIN PEN CAP) WITH CASE REPORT.

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Cleveland, Ohio.

The literature is voluminous with reports of many varieties of foreign bodies in the food and air passages. It is extremely rare to encounter a tracheal foreign body as large as a fountain pen cap.

Jackson first called attention to the fact that each foreign body encountered presents its own problems. The following case report will further illustrate this point.

CASE REPORT.

T. T., a 12-year-old female, was brought to the emergency room at St. Luke's Hospital about 11:30 P.M. on Oct. 1, 1949, approximately one and one-half hours after she had accidentally choked on the cap of a fountain pen. She had been rushed to a neighborhood clinic where an X-ray demonstrated the fountain pen cap apparently in the trachea at the level of the bifurcation. Since she was not having any respiratory distress she was brought to St. Luke's Hospital where adequate bronchoscopic equipment was available.

Family and past history was noncontributory.

Physical examination was normal except for distant breath sounds over the left lung fields. There was no dyspnea or cyanosis.

The X-ray brought with the patient showed a fountain pen cap lodged at the level of the bifurcation of the trachea with the open end up. In order to more conclusively localize the foreign body, the patient was taken to X-ray for a lateral view of the chest. Within a few seconds after the film was taken, the patient suddenly began to struggle for air, became very dyspneic and cyanotic. She was taken to surgery where her color improved with the inhalation of oxygen.

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A small amount of intravenous sodium pentothal was administered and as soon as relaxation of the jaw muscles was obtained, a 7 mm. Jackson bronchoscope was inserted directly, through the cords and into the trachea. The foreign body was found wedged into the opening of the left main stem bronchus with the open end tightly against the right

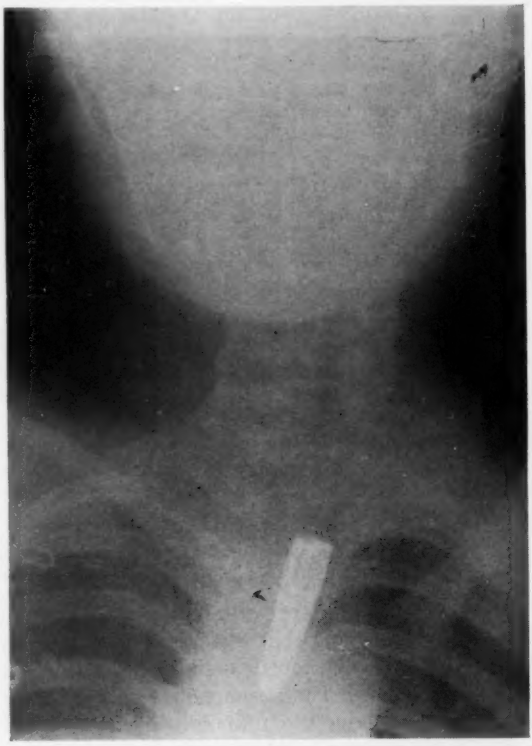

Fig. 1.

lateral wall of the trachea above the bifurcation. The open edge of the cap was grasped with forward grasping forceps, freed from its lodging and brought up into the trachea. The open end of the cap had too great a diameter to protect it with the beveled end of the bronchoscope. The patient's condition necessitated immediate removal of the obstruction, so the cap had to be raked past the cords in order to remove it. The

larynx was examined immediately afterwards. There was a small amount of bleeding in the region of the right cord and considerable congestion and beginning edema. It was deemed advisable to do an immediate tracheotomy. This was done under local 1 per cent procaine anesthesia. A No. 6 tube was inserted; the wound was partially closed with silk sutures to the skin, and the patient was returned to the room in good condition.

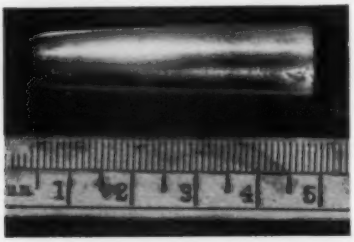

Fig. 2.

Eight hours later the patient could barely squeak any air through the larynx. The tube was blocked off on the fourth postoperative day and was removed on the fifth postoperative day. Secondary closure of the wound was done two days later.

Laryngoscopy one week following the procedure revealed a normal larynx. The voice was completely normal. The patient was discharged from the hospital on Oct. 10, 1949. Indirect laryngoscopy one month later revealed a normal larynx.

COMMENT.

There are two factors of special interest brought out by this case. First, it is surprising that so large a foreign body could lodge in the trachea without causing immediate signs of respiratory obstruction. The absence of a severe cough reflex is probably explained by the immobilization of the foreign body as it became wedged in place just above the bifurcation. The sudden onset of dyspnea and cyanosis can be explained. This occurred when the gradual swelling and edema, caused by the foreign body, suddenly reduced the amount of air which could bypass the foreign body.

The second point worthy of discussion is the method of choice in removing such a large foreign body. It seems logical

that removal of the foreign body through a tracheotomy opening would certainly be the procedure of choice. This would avoid any possibility of laryngeal trauma; however, to use this method, it is necessary to do it before the patient's condition becomes so critical because of severe respiratory obstruction. Again we see the importance of time.

SUMMARY.

1. A case of an extremely large foreign body (metal fountain pen cap) of the trachea is presented.

2. Removal of the foreign body by bronchoscopy caused laryngeal trauma.

3. There may be a delay before onset of severe obstructive symptoms.

4. One should, therefore, consider the presence of a large foreign body in the trachea to be an immediate emergency.

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2. CLERF, LOUIS H.: Foreign Bodies in the Air and Food Passages; Observation on End-Results in a Series of 950 Cases. *Surg., Gynec. and Obst.*, 70:328-339, 1940.

3. JACKSON, C. L.: Endoscopy for Foreign Body. *Ann. Otol., Rhinol. and Laryngol.*, 45:644-654, 1936.

In Memoriam

RAOUL CAUSSE, M.D.,
1892-1949.

It is with great regret that we learned of the passing, on Nov. 16, 1949, of Dr. Raoul Caussé, Director of the Laboratory of Experimental Otology at l'Ecole des Hautes Etudes in Paris. In addition to holding this position, Dr. Caussé was a laureate member of the French Academy of Science. He was also the founder and, for many years, president of the French Society of Otolaryngology and editor of the *Annales d'Oto-Laryngologie*. He was consulting physician of the French Air Force and professor at l'Ecole nationale superieure des Telecommunications. It is unfortunate that Dr. Caussé's work was not better known by American otolaryngologists, physiologists and audiologists. He possessed the rare combination of facility in disciplined research and sensitivity to the needs of clinicians. His more than 200 publications include work on cochlear microphonics and action potentials, electroacoustics, the effects of noise on animals, postgalvanic nystagmus and the toxic action of streptomycin on the vestibular system. He developed many unique histological techniques and always insisted on histological controls in all of those experiments where it was possible.

It was my pleasure to visit Dr. Caussé in his laboratory last Spring. I was impressed by his versatility, not only as an intelligent clinician, but also as a physiologist and mathematician. He spoke of looking forward to a trip to the United States to acquaint himself with progress in otolaryngology, audiology and the neurophysiology of the auditory system.

Dr. Caussé is survived by his widow and daughter, both of whom are physicians. His son-in-law is also an otolaryngologist and it is hoped that he will continue the fine work of his father-in-law.

In the perspective of time Dr. Raoul Caussé will loom as a great figure in otolaryngology. S. R. SILVERMAN.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

There will be a joint three-day meeting, Sept. 11, 12 and 13, 1950, of the North Carolina Society Eye, Ear, Nose and Throat and the South Carolina Society of Ophthalmology and Otolaryngology. Headquarters will be the Skyland Hotel, Hendersonville, N. C.

The following otolaryngologists will be on the program:

Dr. Fletcher D. Woodward, University of Virginia School of Medicine, Charlottesville, Va.

Dr. Lester A. Brown, Emory University School of Medicine, Atlanta, Ga.

Dr. B. T. Horton, Mayo Clinic, Rochester, Minn.

Dr. Donald F. Proctor, Johns Hopkins University School of Medicine, Baltimore, Md.

Ophthalmologists to be present are:

Dr. Bruce Fralick, University of Michigan School of Medicine, Ann Arbor, Mich.

Dr. Edmund B. Spaeth, University of Pennsylvania School of Medicine, Philadelphia, Pa.

Dr. Frank Walsh, Johns Hopkins University School of Medicine, Baltmore, Md.

Dr. Richard G. Scobee, Washington University School of Medicine, St. Louis, Mo.

For any further information, please contact: Dr. MacLean B. Leath, Secretary, North Carolina Society Eye, Ear, Nose and Throat, High Point, N. C., or Dr. Roderick Macdonald, Secretary, South Carolina Society of Ophthalmology and Otolaryngology, Rock Hill, S. C.

INTERNATIONAL COURSE IN AUDIOLOGY, STOCKHOLM, SEPTEMBER, 1950.

In a letter to the editor, published in Vol. 37, No. 6, of *Acta Oto-Laryngologica*, it was proposed that a practical course in audiology should be arranged in Stockholm this year.

In the editor's reply the desirability of arranging such a course was emphasized, and all those who might be interested were requested to communicate with *Acta Oto-Laryngologica*. The response was most encouraging, and the large number of inquiries and preliminary enrollments attest the very keen interest that has been aroused by the proposal. The signatories of this communication have therefore formed a committee for the purpose of organizing such a course, to run from the 11th to the 20th of September.

The following lectures will be given:

A. *Lectures:*

I. Anatomical, Physiological, Physical and Psychological Backgrounds.

II. Hearing Loss—its prophylaxis, incidence, causes and psychology.

III. Hearing Tests. *1.* Physical Basis of Audiometry. *2.* Comparison between old and modern methods. *3.* Tone Audiometry — basic and special tests — interpretation. *4.* Speech Audiometry—basic and special tests—interpretation. *5.* Group Audiometry—basic and special tests—interpretation. *6.* Standardization of Determinations, Audiometers and Audiograms.

IV. Treatment of Hearing Loss. *1.* Treatment of Hearing Loss in Children. *2.* Medical and Surgical Treatment of Hearing Loss, Except in Otosclerosis. *3.* Fenestration.

V. Remedies. *1.* Speech Re-education. *2.* Lip Reading. *3.* Hearing Aids. *4.* Rehabilitation and Re-employment. *5.* Social, Psychological and Educational Aspects. *6.* The Audiology Center.

B. Practical Courses and Demonstrations:

I. Tone and Speech Audiometry—Basic and Special Test Methods.

II. Selection of Hearing Aids.

III. Fenestration: training on specimens . (The course will be arranged if the amount of interest warrants it. Reservations can be made only for a few participants. A special fee will be payable to cover the costs of specimens, instruments, etc.).

The above survey is not to be regarded as the final program for the course. Broadly speaking, it is proposed to give the theoretical training in the forenoons, with two or three lectures for the entire assembly, and the practical training for two or three hours in the afternoons to small groups of participants.

For further information, address C. A. Tegnér, Secretary, 33 Birger Jarlsgatan, Stockholm C. Sweden.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of February 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc. 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.